CLINICAL PROFILE AND OUTCOME OF PANCREATITIS IN CHILDREN AGED LESS THAN 15 YEARS

Dissertation Submitted to

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY
CHENNAI, TAMILNADU

In fulfillment of the regulations for the award of the degree

M.D.(PEDIATRICS)

DEPARTMENT OF PEDIATRICS

PSG INSTITUTE OF MEDICAL SCIENCES & RESEARCH

COIMBATORE, TAMILNADU

APRIL 2016

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M.D. (PEDIATRICS)

GUIDE

DR. JOHN MATTHAI

DEPARTMENT OF PEDIATRICS

PSG INSTITUTE OF MEDICAL SCIENCES & RESEARCH

THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY
CHENNAI, TAMILNADU

APRIL 2016

DECLARATION

I hereby declare that this dissertation entitled **"CLINICAL PROFILE AND OUTCOME OF PANCREATITIS IN CHILDREN AGED LESS THAN 15 YEARS"** was prepared by me under the guidance and supervision of **Dr. JOHN MATTHAI**, Professor and Head of the Department of Pediatrics, PSGIMS&R, Coimbatore.

This dissertation is submitted to The Tamilnadu Dr. M.G.R Medical University, Chennai in fulfillment of the university regulations for the award of MD degree in Pediatrics. This dissertation has not been submitted elsewhere for the award of any other Degree or Diploma.

Dr.SENTHIL AAKASH. K

CERTIFICATE

This is to certify that the thesis entitled **"CLINICAL PROFILE AND OUTCOME OF PANCREATITIS IN CHILDREN AGED LESS THAN 15 YEARS"** is the bonafide work of **Dr.SENTHIL AAKASH. K**, done under my guidance and supervision in the Department of Pediatrics, PSG IMS&R, Coimbatore in fulfillment of the regulations laid down by The Tamilnadu Dr. M.G.R. Medical University for the award of MD degree in Pediatrics.

Dr. JOHN MATTHAI
Professor and
Head of the Department
Department of Pediatrics
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"Clinical profile and outcome of pancreatitis in children aged less than 15 years"

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2. Proposal
3. Assent Forms
4. Parental Consent Forms
5. Confidentiality Statement
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The approval is valid for one year.

We request you to intimate the date of initiation of the study to IHEC, PSG IMS&R and also, after completion of the project, please submit completion report to IHEC.

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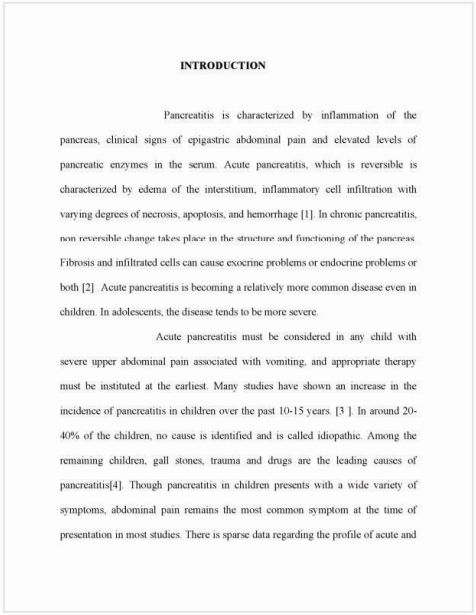
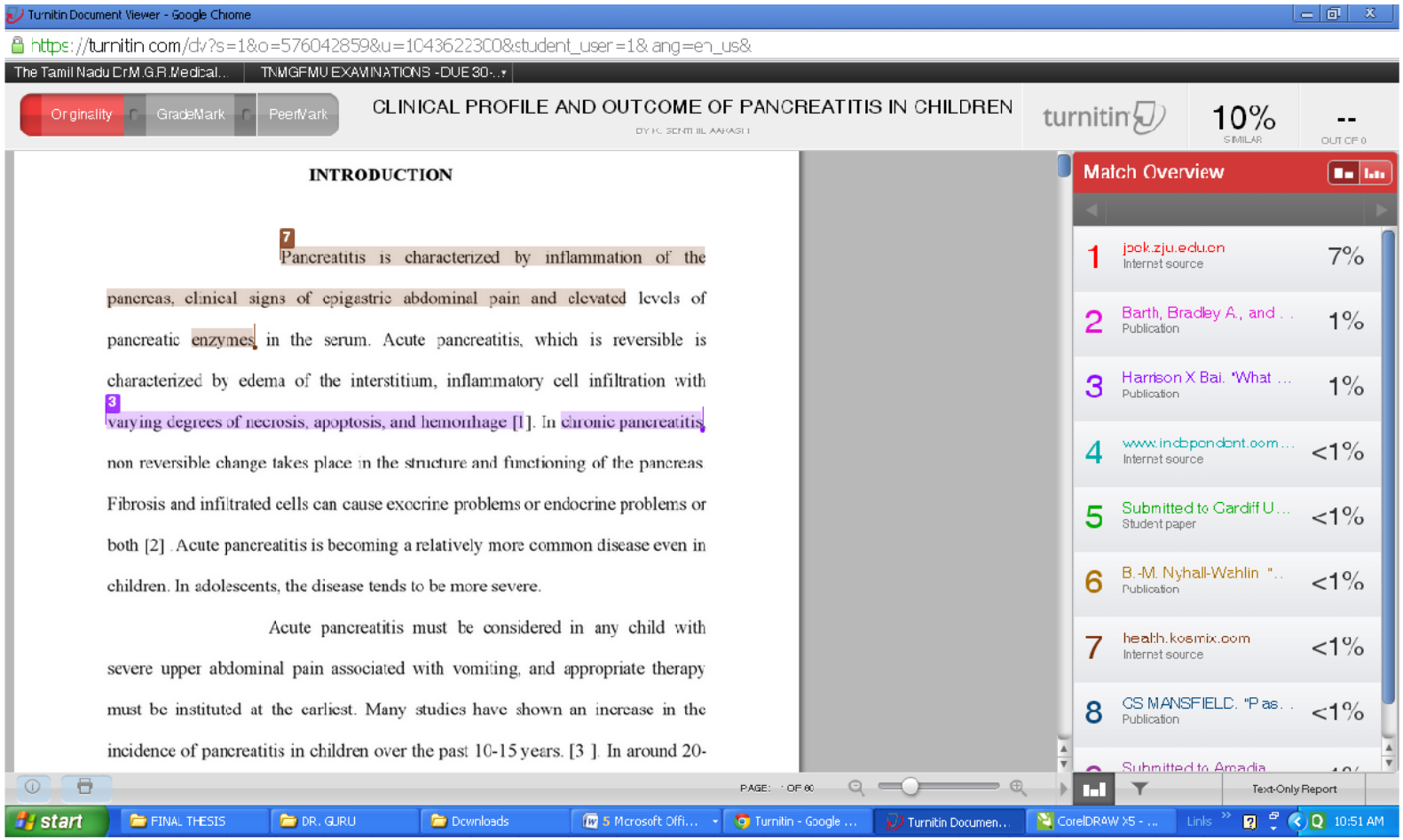


INTRODUCTION

Pancreatitis is characterized by inflammation of the pancreas, clinical signs of epigastric abdominal pain and elevated levels of pancreatic enzymes in the serum. Acute pancreatitis, which is reversible is characterized by edema of the interstitium, inflammatory cell infiltration with varying degrees of necrosis, apoptosis, and hemorrhage [1]. In chronic pancreatitis, non reversible change takes place in the structure and functioning of the pancreas. Fibrosis and infiltrated cells can cause exocrine problems or endocrine problems or both [2]. Acute pancreatitis is becoming a relatively more common disease even in children. In adolescents, the disease tends to be more severe.

Acute pancreatitis must be considered in any child with severe upper abdominal pain associated with vomiting, and appropriate therapy must be instituted at the earliest. Many studies have shown an increase in the incidence of pancreatitis in children over the past 10-15 years. [3]. In around 20-40% of the children, no cause is identified and is called idiopathic. Among the remaining children, gall stones, trauma and drugs are the leading causes of pancreatitis[4]. Though pancreatitis in children presents with a wide variety of symptoms, abdominal pain remains the most common symptom at the time of presentation in most studies. There is sparse data regarding the profile of acute and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INTRODUCTION

Pancreatitis is characterized by inflammation of the pancreas, clinical signs of epigastric abdominal pain and elevated levels of pancreatic enzymes in the serum. Acute pancreatitis, which is reversible is characterized by edema of the interstitium, inflammatory cell infiltration with varying degrees of necrosis, apoptosis, and hemorrhage [1]. In chronic pancreatitis, non reversible change takes place in the structure and functioning of the pancreas. Fibrosis and infiltrated cells can cause exocrine problems or endocrine problems or both [2]. Acute pancreatitis is becoming a relatively more common disease even in children. In adolescents, the disease tends to be more severe.

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There is sparse data regarding the profile of acute and chronic pancreatitis in children particularly from the developing countries. This study was undertaken to profile the clinical features, etiology and outcome of acute and chronic pancreatitis in children.

OBJECTIVES

- **Primary Objective:**

  To study the etiology, presentation and management of pancreatitis in children less than 15 years.

- **Secondary Objective:**

  To determine the complication and recurrence rate of pancreatitis in children.

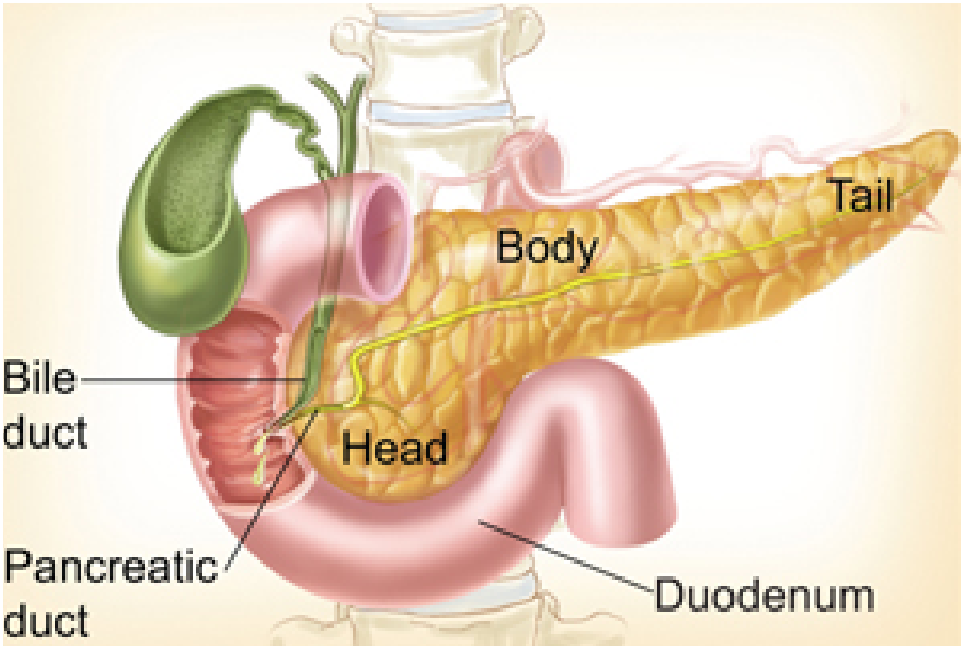
REVIEW OF LITERATURE

History :

Pancreas was referred as "finger of the liver" in Talmud, which was mentioned many years back. Vesalius thought that pancreas was a cushion for the stomach. Galen realised that pancreas will give protection and support to the vessels in the body. Wirsung explained about the ducts within the pancreas of human beings in 1642 . de Graaf explained about the secretion of the gland pancreas from the fistula of pancreas of dogs. The digestive activity of pancreatic gland was demonstrated much later. Later, Eberle and Purkinje and explained about pancreatic activity of emulsifying fat, proteolysis activity, and digesting activity by pancreatic enzyme secretions. Subsequently, Bernard described the digesting activity of pancreatic secretions on lipids, carbohydrates and proteins. In the year of 1876, Kuhne coined the term as enzyme and extracted trypsin . In 1889, Chepovalnikoff, discovered enterokinase in the mucosa of the duodenum. It is required for activating the proteolytic enzymes. In 1895, Dolnsky stimulated the secretions in the pancreas by pouring acid into the duodenal mucosa. These things made the discovery of the hormone secretin by Baylis. It is the first hormone to be identified. In 1869, Langerhans described the histologic structure of the pancreas. Shortly After that, Hidenhain demonstrated changes in the postprandial state that happened in the dog. Friedreich was the first person who described pancreas systematically in 1875.

ANATOMY

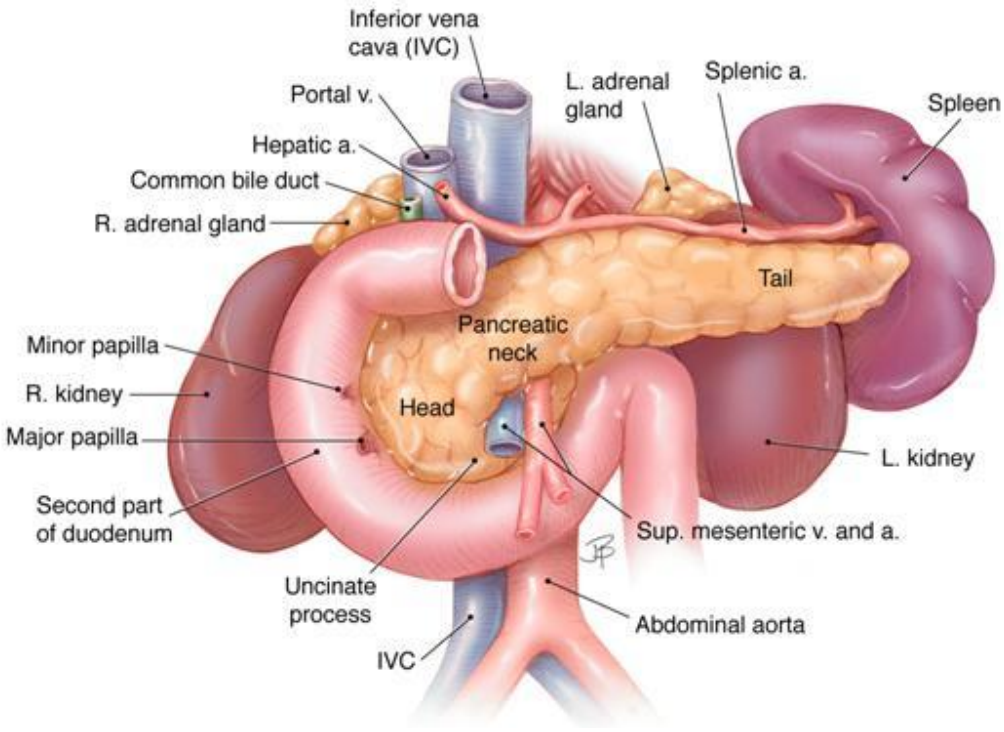
Figure 1: Structure of Pancreas



The pancreatic gland is soft, which is slightly flattened and also elongated [4-6]. The head of pancreas lies underneath the peritoneal layer of the posterior wall of the abdomen and it has a structure resembling that of a lobule. The pancreatic gland does not have a true capsule. It is covered with a fine connective tissue. The pancreatic head lies on the right end and is placed in the curve of the duodenum. The body of pancreas, neck, and the pancreatic tail lie in an oblique fashion in the posterior part of the abdomen. The tail of pancreatic organ reaches till the gastric side of the spleen. The 2nd and 3rd curvatures of the duodenum lie encircling the pancreatic head. The anterior surface of the pancreatic head lies adjacent to the pyloric region of stomach, the 1st part of

duodenum, and transverse part of the colon. The posterior surface touches the hilum and medial surface of right kidney, the right gonadal vein, the right renal vessels and the inferior vena cava. The uncinate process of pancreas is usally a prolonged part of the pancreas with different shapes and size. It usually extends from the lower portion of head, and extends in upward direction and to left side. The uncinate process of pancreas lies just anterior to inferior vena cava and the aorta. The uncinate process is covered above by the superior mesentric vessel. The uncinate process may also not seen.

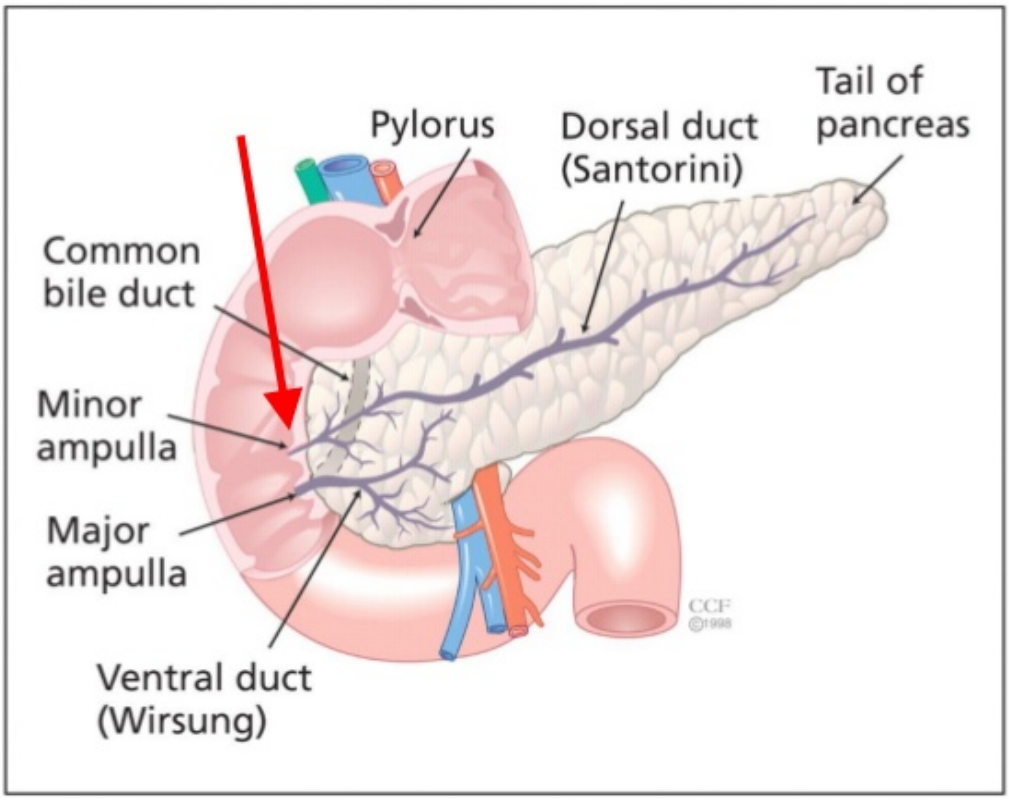
Figure 2: Relations of Pancreas



The pancreatic neck appears to be constricted and it extends from the head towards the left, joining with the body of the pancreas. The joining of the portal vein with superior mesenteric vein and splenic vein lies posterior side of the neck of pancreas . Anteriorly, the neck of pancreas is enveloped by pyloric part of stomach and peritoneal layer of lesser sac. The body of the pancreas lies infront of the major blood vessel aorta. It lies behind the peritoneam and is held towards the aorta by the lesser sac peritoneum. The anterior surface of the body of pancreas is covered by peritoneal layer of the omental bursa. The antrum portion and body part of the stomach had contact with the body of pancreas on the anterior side. Posteriorly, body of the pancreas is related to the aorta, the origin of the SMA, the left kidney, the left adrenal gland, the left crus of the diaphragm. The middle of the body of pancreas lies over the lumbar spine. This part is mostly injured in case of any trauma to abdomen. Between the body and the tail, there is no junction point. The tail of the pancreas is mobile, and its tip reaches the splenic hilum. The tail of pancreas lies between the two layers of the splenorenal ligament.

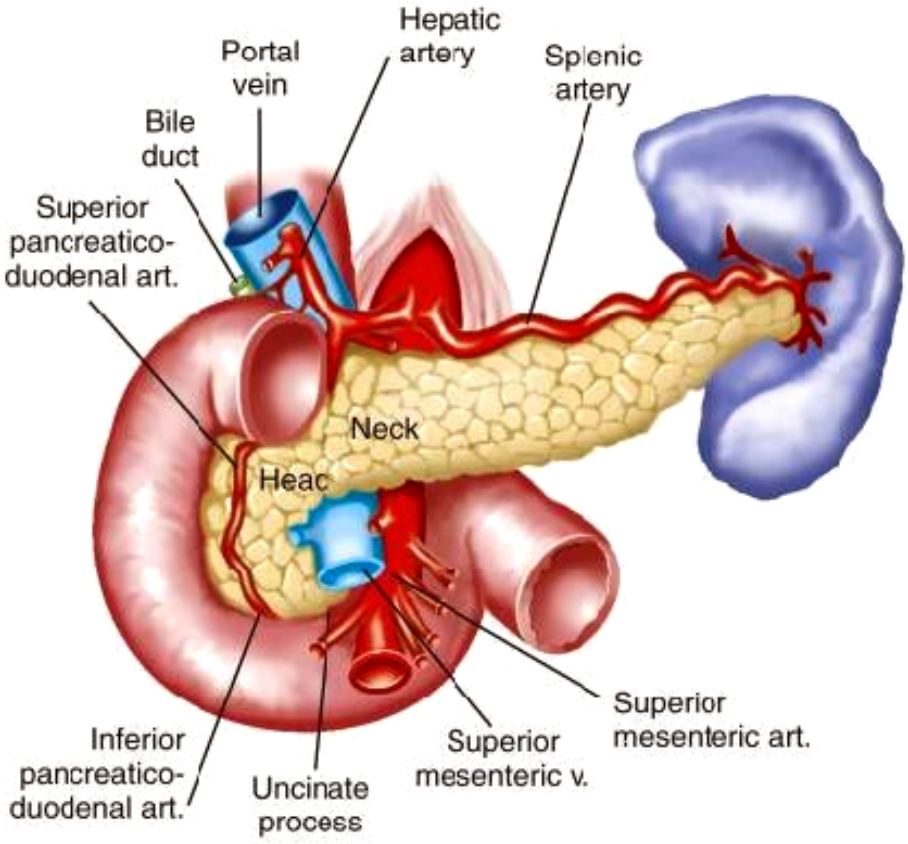
The duct of Wirsung originates near the tail end of the pancreas. It is formed by joining the ductules which drain the lobules. It usually drains into major duodnal papilla. The accesory duct (duct of santorni) usually joins with the main duct and it drains into minor papilla [7].

Figure 3: Ducts of Pancreas



CIRCULATION

Figure 4 : Arterial supply of Pancreas



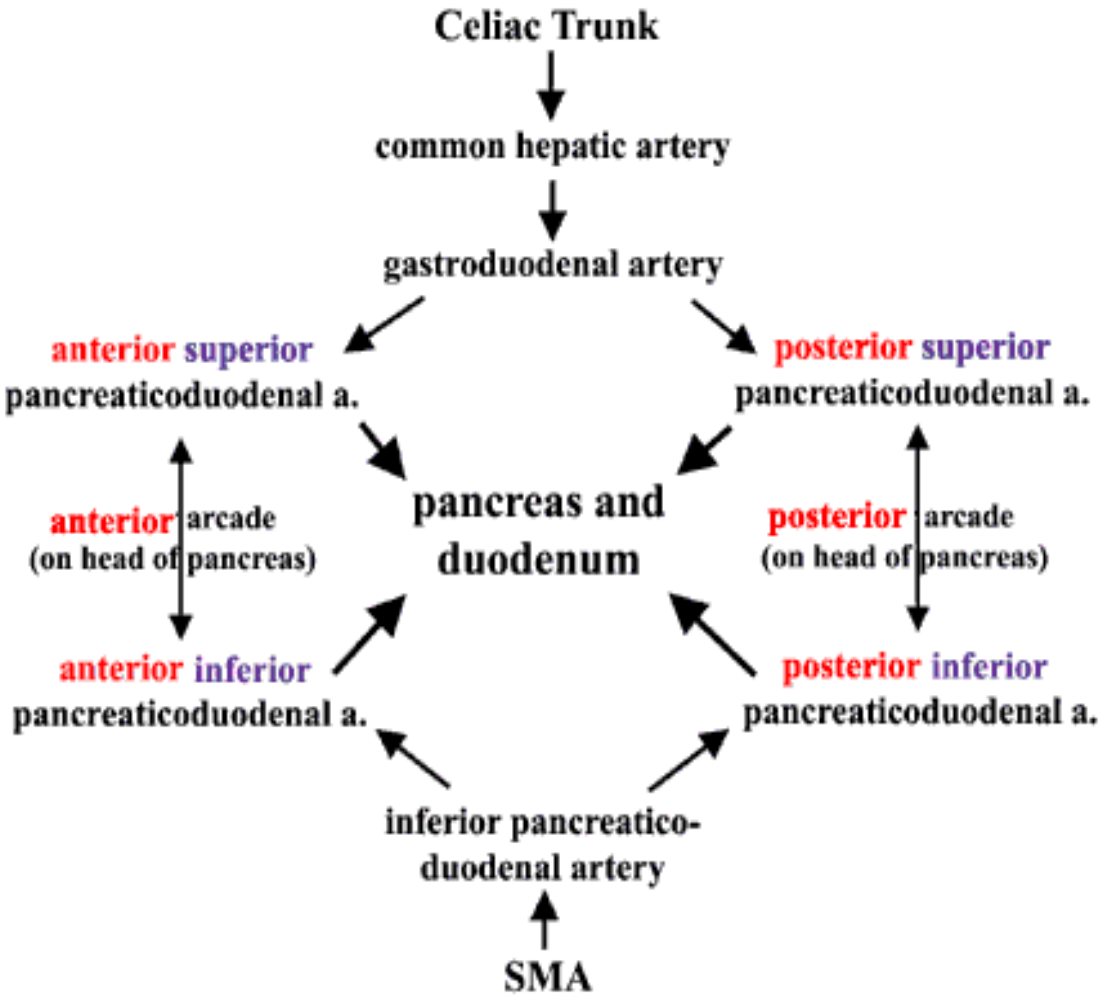
The pancreatic gland is well circulated and it is supplied by branches of the celiac artery and SMA [8,9]. The pancreatic head is supplied by two pancreatcoduodenal arterial arcades. They are formed by the anterior and postero superior pancreatcoduodenal arteries that join with a second pair of anterior and posterior inferior pancreatcoduodenal arteries. The gastroduodenl artery arises from the hepatic branch of the celiac artery. It divides into anterior and posterior superior pancreatcoduodenal arteries. The antero superior

pancreaticoduodenal artery lies on the surface of the pancreatic gland. It gives its branches to the anterior part of the duodenum, proximal part of the jejunum, and pancreas. The artery then enter into the pancreas and, on the posterior side, join with the anteroinferior pancreatcoduodenal artery from the SMA. The anteroinferior pancreatcoduodenal artery arise from the SMA at the inferior end of the pancreatic neck.

The posterinferior pancreatcoduodenal artery arises from the gastrduodenal artery. Its course is there on the posterior side of pancreas, and may join with branches of the gastroduodnal artery or with a branch from dorsal pancretic artery. It passes posteriorly to the pancreatic portion of the bile duct. In the neck of pancreas, the dorsl pancretic artery arises from the splenic artery. From here, a right branch usually gives blood supply to the head and joins the posterior vessels. It also supplies and then gives branches that carry through the body of the pancreas and tail, often having connections with some of the branches of the spleenic artery and a more distal end connection with the spleenic or the left gastrepiploc artery.

All other major arteries usually lie posterior to the ducts. The course of the spleenic artery lies posterior to the distal portion of pancreas and it encircles above and below the upper margin of the pancreatic gland. The dorsal pancretic artery, joins with one of the posterior arcades after branching the inferor pancretic artery. The caudal pancretic artery usually get arised from the L.

gastroepiploc artery or from a splenic branch . It then joins with branches of the spleenic and some great pancretic arteries and with other pancreatic arteries. (Figure 4)



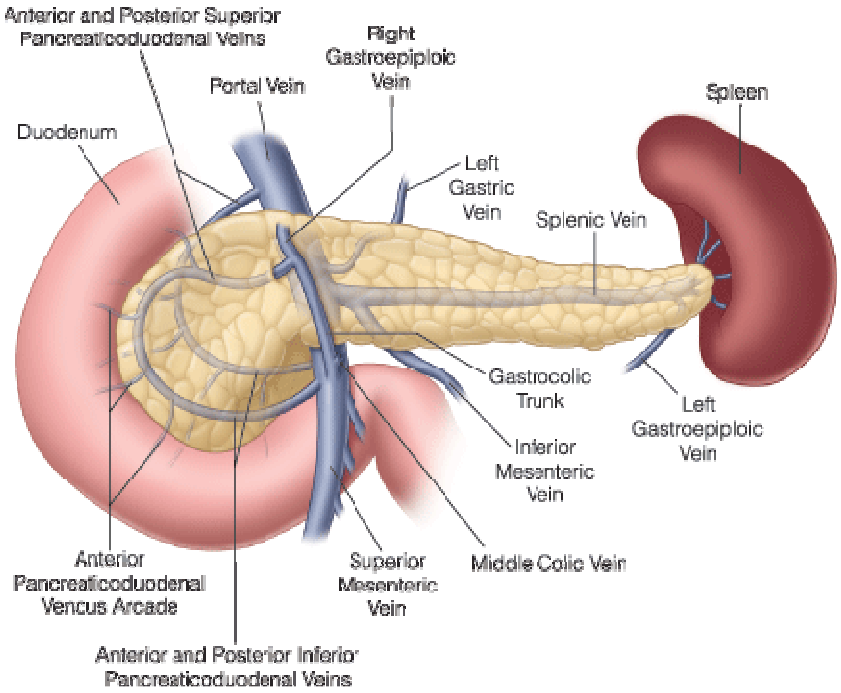


Figure 5: Venous drainage of the pancreas

Generally, the venous system of pancreas is like that of the arterial supply. It drains into the portal system, and the system which is mainly draining the pancreas is formed by the combination of the superor mesenteric vein and the splenic veins behind the neck part of the pancreas. The portal vein gets behind the pancreas gland and is situated to the front of the IVC, with the bile duct to right and on the left side by the hepatic artery. The spleenic vein starts at the hilum part of the spleen and usually forms a curve behind the tail portion of the pancreas and underneath the spleenic artery, to the right side along the posterior region of the pancreatic gland. The pancreatic veins originates from the the neck of the pancreas,its body, and tail portion of the pancreas and join the splenic vein. The pancreticoduodenal veins run very close proximity to their pancreatic

arteries and drain into the spleenic or portal veins. Due to the close proximity of the splenic vein with pancreatic gland,any inflammation or neoplasm involving the pancreas can lead to occlusion of the splenic vein. Due to this, there is backward flow toward the spleenic hilum and then, through the gastric and left gastroepiploc veins, can cause gastric varices. (Figure 5)

LYMPHATIC DRAINAGE

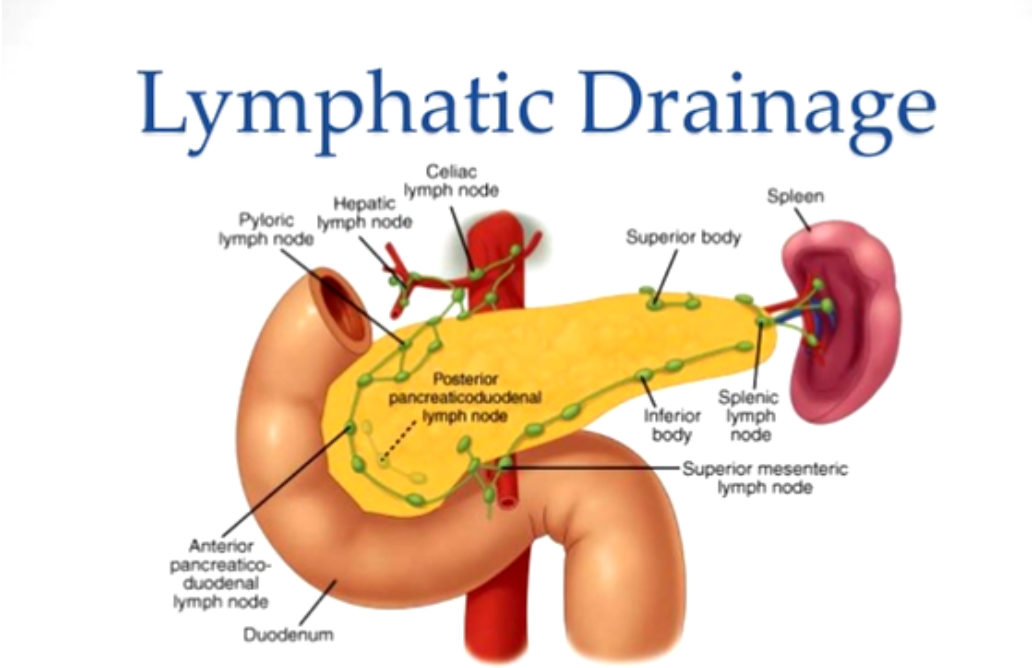


Figure 6 : Lymphatic drainage of pancreas

The lymphatics in the pancreas drain the surface and it carries the lymph toward regional nodes which lie near the large blood vessels[10,11]. The superior group of lymphatics pass along the upper end of the pancreatic gland in close proximity with the spleenic vessels. Those lymphatics on the left side of

the pancreas drain into nodes in the spleenic hilum. Those lymphatics on the right side and near to the body and the neck empty into lymph nodes near the upper end of the head. They also receive lymphatic drainage from the anterior surface and posterior pancreatic surfaces. The inferor lymphatic vessel run with the inferor pancretic artery. Those that drain the lower left side of the body and tail drain toward nodes in the splenic hilum. The remaining portions of the neck and body drain toward the right. Lymphatic vessels drainage of the head of the pancreas is broadly divided into an anterior lymphatic system and a posterior lymphatic system. These vessels usually lie in the grooves in between the head and the duodnum, near the pancreatcoduodenal blood vessels. Each group of drainage system (i.e anterior group and posterior) also has subgroups- superior and inferior type of drainage systems. In addition to this, a separate set of lymphatics vessels also drains the upper part of the head, which lies on the superior border. The lymphatics of the head of pancreas and duodenal part flows into the celiac group and superior mesenterc groups of pancretic nodes and then drainsinto the cisterna chyli.

The lymphatic supply of the tail of the pancreas drain into spleenic hilar nodes and the lymphatics of the body of the pancreas pass to the pancreatcosplenic nodes which lies alongwith the superior border,and then it drains into the coeliac nodes. Lymphatic drainage of the upper part of head of the pancreatic gland pass through subpyloric group of nodes. In the inferior

portion, lymphatics drain into the retropancretic and antepancretic nodes, which then joins and then drains into superior mesenteric group of nodes. (Figure 6)

INNERVATION

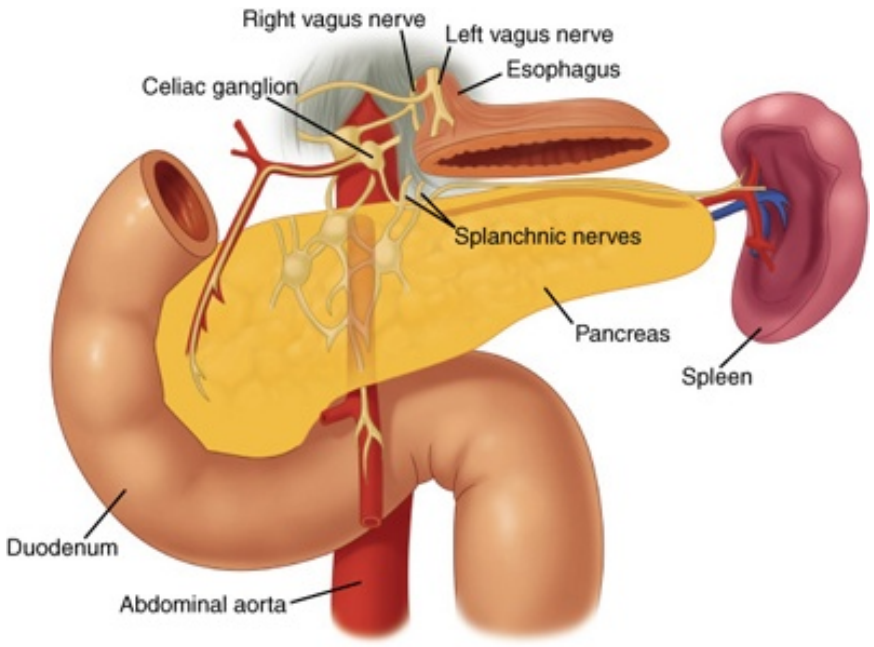


Figure 7 : Nerve supply of pancreas

The visceral main efferent nerve supply of the pancreas is by the vagus and splanchnc nerves by the hepatic nerve plexus and coeliac plexuses. The efferent nerves of the vagus does not synapse and pass through and end in parasympathetic ganglia which is there in the intrlobular septa of the pancreatic gland. The postganglionc nerve fibers gives nerve supply to pancreatic acini, islet cells, and the ducts. The neurons of the sympathrtic efferent nerves begins in the lateral part of the grey matter of the thoracic part and lumbar part of the

spinal cord. The nerve bodies of the postganglionc sympathetc neurons are situated in the great plexus within the abdomen. Their postganglionc fibers provides nerve supply to only blood vessels. The autonomc fibers are situated in close proximity to the blood vessels of pancreas. Nothing much is known regarding the distribution of the visceral group efferent fibers in humans. They mostly run through the splanchnc nerves to the sympathatic trunks and rami communictes and through spinal nerves and through ganglia. The vagus also carry some visceral efferent fibers. (Figure 7)

HISTOLOGY AND ULTRASTRUCTURE:

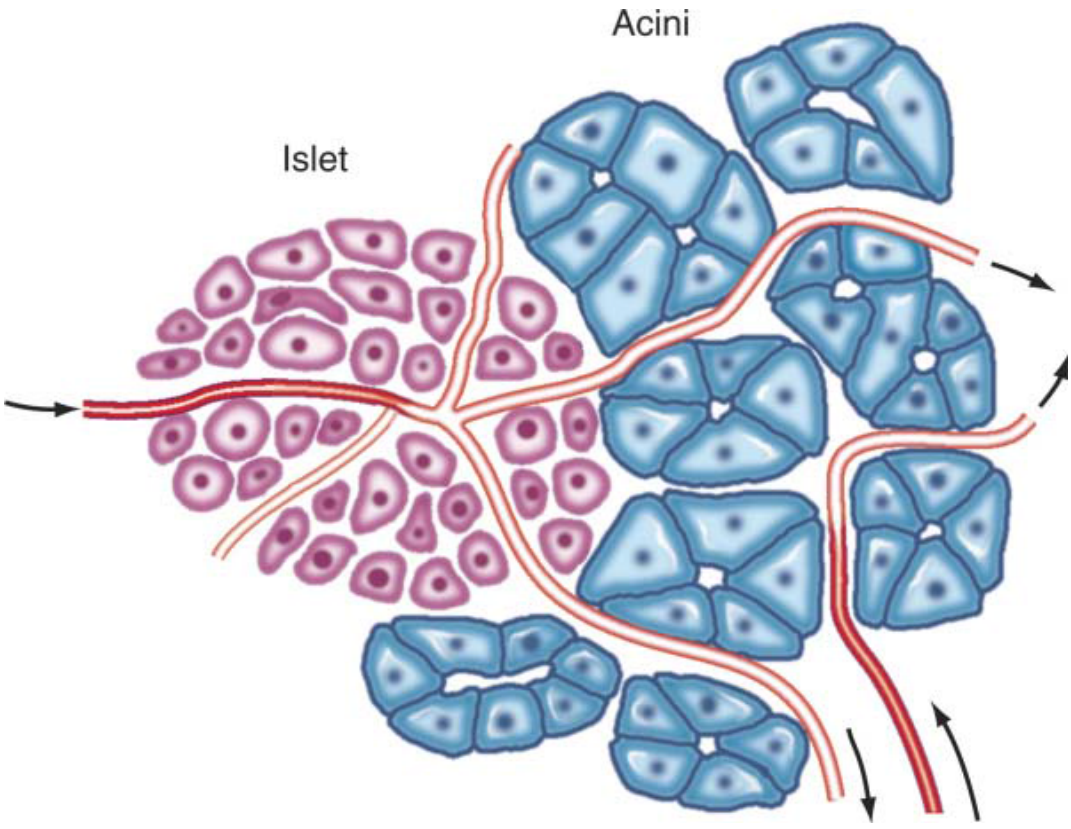


Figure 8 : Ultrastructure of the pancreas

The pancreatic gland is a compound structure, which is nodular that is similar in structure to salivary glands but it is less compact than that. Though connective tissue surrounds it, there is no fibrous capsule. The lobules within the gland are visible on examination and connective tissue is present which connects the lobules and it embeds the vessels, excretory ducts and the nerves. As it is a mixed gland, it contains both exocrine cells which is in majority and endocrine cells in less number. The endocrine part of the gland which is less in number consists of the Langerhns cells. They are rounded cells which are in clusters and are present throughout the pancreatic gland.

The exocrine part of the pancreas has many acini which is composed of tubular mass and rounded masses of cells, and they are the subunits of the lobule[12,13].(Figure 8). Silicon type of casts in the lumen of the duct formed by retrograde injection generally indicate that the acini are more in number and the cells are mainly curved, branching type of tubules that get anastomosed and ends blindly . The secretory duct originates from the acini lumen and it contains centroacnar cells, and these cells are unique to pancreas. The centroacinar cells stain pale in histological sections and relatively smaller than the acinr cells. Columnar epithelial cells covers the intralobular ducts and the acini lumen gets into the ducts . These intralobular ducts are not striated and they anastomose with each other to form the interlobulr ducts, and columnar type of epithelium lines this. Goblt and occasional argentafin cells also are seen. The interlobulr

ducts anastomose with each other to form the main pancreatc duct. Connective tissue and elastic fibers are present in large ducts. There is a central lumen with a broad base and the acinar cells are lined by columnar epithelial cells. Many number of eosinophilc zymogen granuls present in the apical part of the cell in the resting state. spherical nuclei and basophilic cytoplasm are present in the cells basal portion. Inbetween the nucleus and granules lies the golgi complex and it is a nonstaining region . After feeding and digestion, the acinar cells usually have cyclical changes in the morphology. After having a large meal, the acinar cells zymogen content is lost. There will be a reduction in both the number and size of granules. (Figure 9)

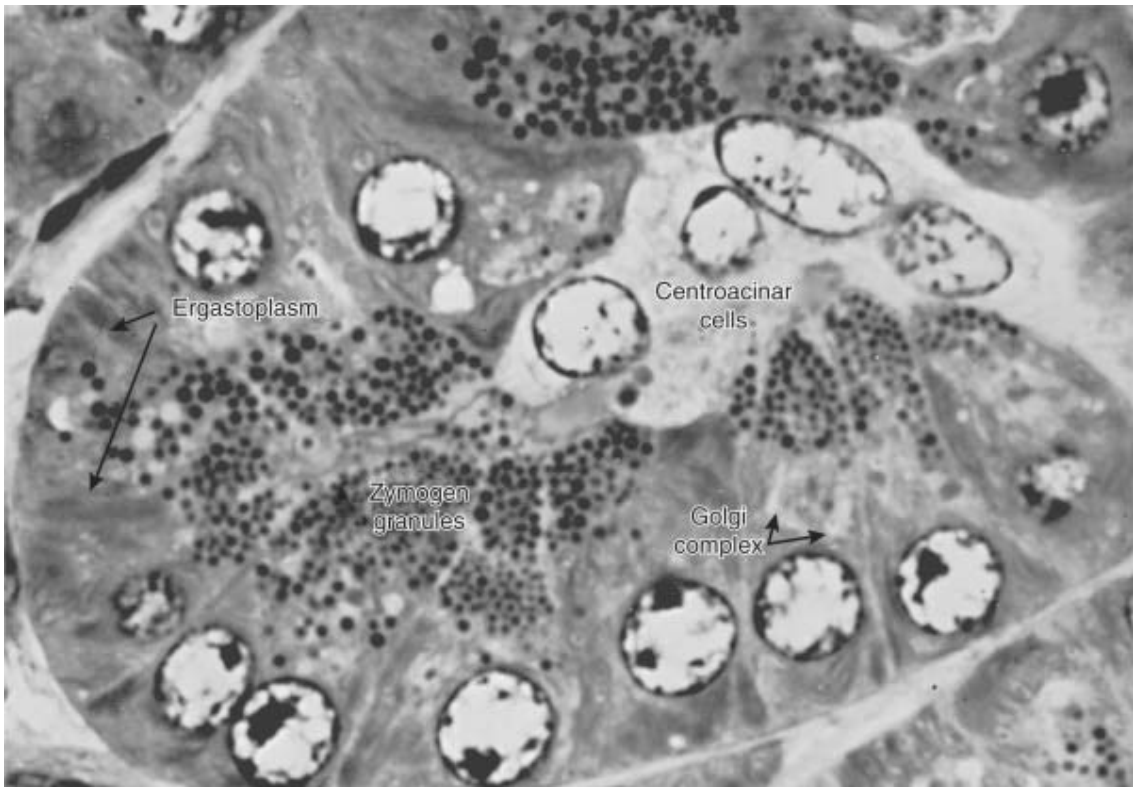
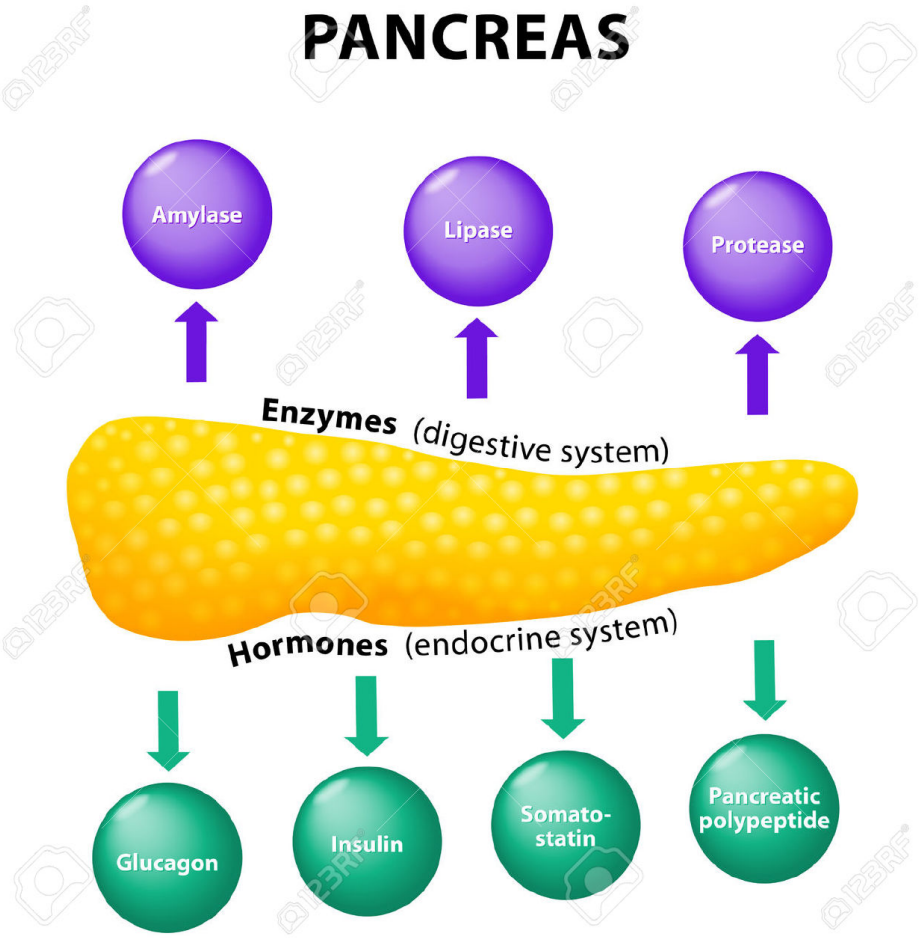


Figure 9 : Histological structure of pancreas

PANCREATIC SECRETIONS:



Pancreatic secretions play a major role for digestion and also function as hormones in the endocrine system. The enzymes secreted by the pancreas which aids in digestion are lipase, amylase and proteases. The hormones secreted by the gland are glucagon, insulin, somatostatin and pancreatic polypeptide

ETIOLOGICAL FACTORS OF ACUTE PANCREATITIS

In adults, most of the pancreatitis episodes are due to cholelithiasis or induced by alcohol, but in childhood pancreatitis, the etiologies are much different. We could not clearly classify the etiology in children due to differences in their prevalence in various studies. This difference in the causative factors is mainly due to retrospective nature in which most of the studies were conducted. As investigations are not complete, this also can further complicate the issues. As nowadays many etiologies are newly recognized, the categories can be splitted into more number. The currently available data regarding pancreatitis suggests that drugs, biliary disease, traumatic causes, systemic disease,and idiopathic followed by metabolic causes, pancreatitis due to infections and hereditary with family history of pancreatitis are the common causes. [3,4,16-19,20-23].(TABLE 1)

TABLE 1 : CAUSES OF PANCREATITIS

CAUSES OF PANCREATITIS IN CHILDREN

ACUTE

Idiopathic

Blunt abdominal trauma

Drugs

Viral infection

Hereditary disorders

Bilipancreatic tract anomalies

Cholestasis

CHRONIC

CALCIFIC	NON CALCIFIC
Juvenile tropical	Congenital/acquired lesion of the duct
Hereditary	Trauma
Errors of metabolism	Sphincter of Oddi dysfunction
Hyperlipidemia	Sclerosing cholangitis
Hypercalcemia	Idiopathic fibrosing
Hyperparathyroidism	Renal failure
Cystic fibrosis	

Biliary tract disease

Gallstones or presence of sludge within the gallbladder was seen in around 20-30% children with pancreatitis.[3,4,16,18,21-25,]. The defects in the structure of pancreas like pancreatic divisum and Oddi sphincter dysfunction are also included under biliary causes. Tumours or stones cause biliary obstruction leading to pancreatitis in the adults but in children biliary sludge constitutes major part [3,4,18,22]. In children, most of them have sludge in the gall bladder and not fully formed stones which leads to this difference in etiology. The child with pancreatitis when caused due to biliary pathology, they have elevated SGPT and SGOT levels with raised serum bilirubin[22]. The stone shall be removed by ERCP in case the obstruction persists for more than 3 days or if cholangtis or pancreatits gets worsened. At present, there is no clear evidence regarding the treatment of sludge but some clinicians used ursodiol for managing these children. But they were not sure that pancreatitis got improved and there is free flow of bile as the obstruction was cleared both occurred in a spontaneous way or due to drugs. Children having biliary stones, surgical removal of gall bladder should be done within 4 weeks as per the guidelines. If the pancreatitis was recurrent only, surgical removal was indicated otherwise it is not needed. (Figure 7)

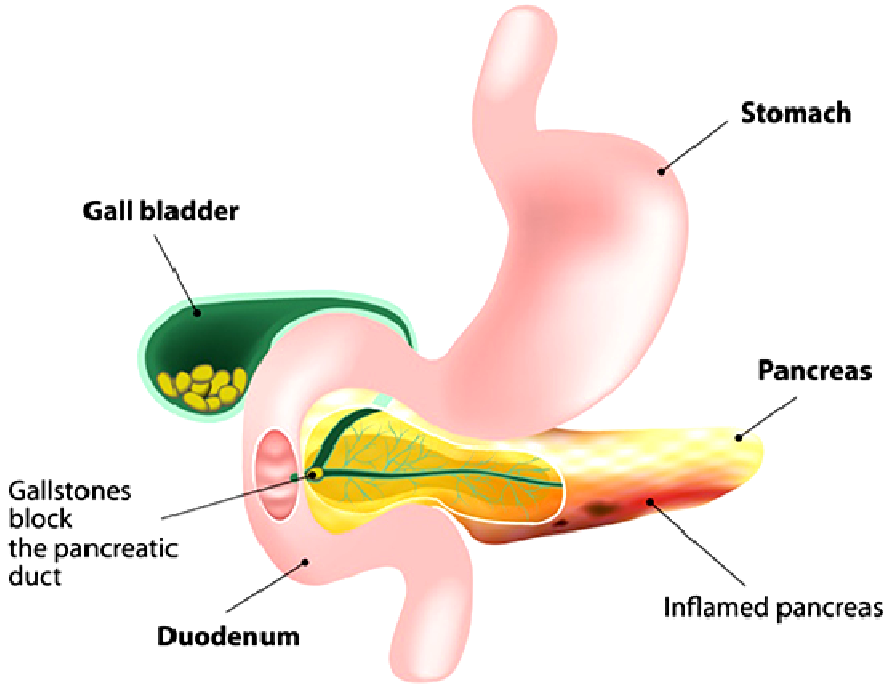


Figure 10 : Gall stones in acute pancreatitis

Pancreatic divisum

It is a congenital anomaly in which instead of a single duct there are 2 ducts. There are 3 types- classical, absent ventral duct and functional.

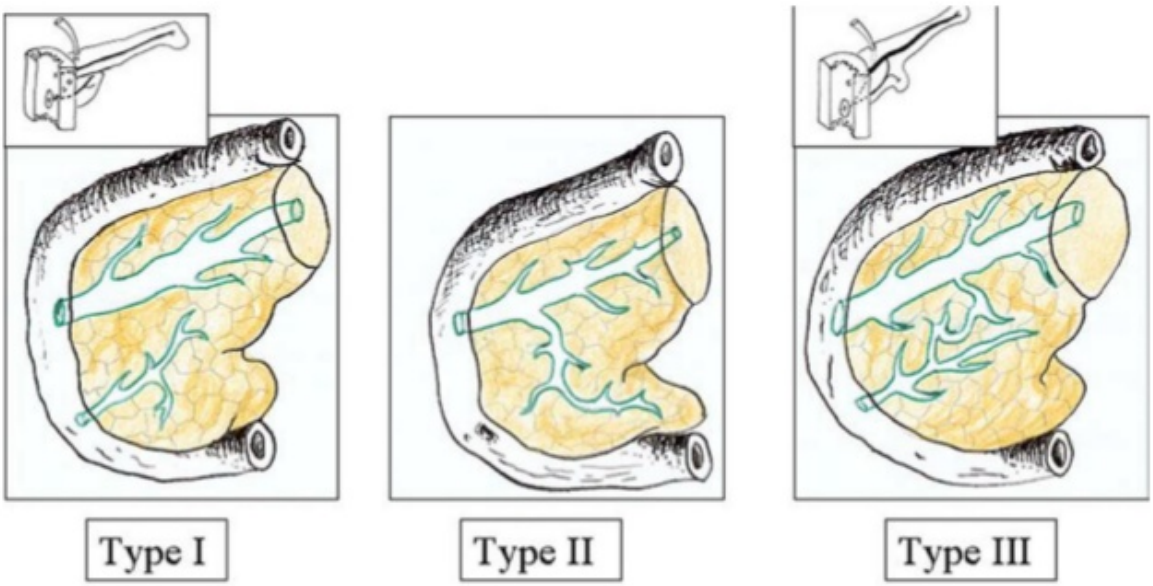


Figure 11 : Types of pancreatic divisum

Drugs:

In only less than one fourth of cases of pancreatitis, drugs were postulated as causing pancreatitis[3,4,15,16,19,21,23,24]. The drugs mainly causing the disease were valproic acid, asparginase and prednisolone. The study persons are not aware of what is the relation between the ingestion of the drug and development of pancreatitis and whether any mechanism was available behind this. Many children who take drugs also have some systemic disorders so we are not aware pancreatitis is caused either by those drugs or by the systemic disorder.

Idiopathic :

Even nowadays despite having many efficient investigations and many detection techniques, idiopathic pancreatitis ranged from 15 to 40% [3,4,17-19,20,22,23,24-26]. So there is no decrease in the incidence of the idiopathic disease despite having these finding modalities.

Systemic disease :

Many systemic associated diseases like cystic fibrosis, HUS, SLE, sepsis and shock can also be a cause of the disease or can be associated with pancreatitis[3,16-18,19,25]. But it was not clear whether the raise in incidence was due to the disease per se or due to raising number of systemic disorders.

Traumatic cause :

Trauma as a cause of pancreatitis was seen in about one third of children. [3,4,15-18,20,22-27]. Children will easily get hurt by playing sports or road traffic accidents or due to any abuse to the child.

Infectious causes:

Pancreatitis caused by infections were seen in less than 10% of the children [3,4,15-18,20,22-27]. In this too, like in drugs and systemic diseases, whether pancreatitis and infections are associated with each other was not well understood. They usually present with symptoms of respiratory tract infections

like fever, coryza etc . Some viral causes which are closely related are varicella [29,38] , mycolasma[30], hepatitis A[32-34] , mumps[22,26,40], rota virus[35-37] and coxsakie virus[39].

Metabolic causes :

The common metabolic derangements which leads to pancreatitis are increase in calcium and triglyceride levels[15,23]. It can be seen in less than 10% of children[3,4,16,17,19,21,23]. These children often presents with recurrent pancreatitis because they often have metabolic derangements which will cause pancreatitis. But the exact reason why metabolic disturbances cause the disease is not well understood. The main modalities of management for these type of children is to correct the underlying disease. For hypertriglycerdemia, lipid reducing agents and statins should be administered. Many children presenting with increased calcium levels may be due to hyprparathyrodism so surgiacal removal of the gland is mandatory for treating these kind of children.

Hereditary cause :

Family history of pancreatitis also plays a significant role in diagnosing the cause of pancreatitis. It was found hereditarily in around 5% children due to various mutations like SPINK and PRSS[3,17,18]. The history of the family

members with detailed history about the past 2 generations and the genetic study are essential ti diagnose hereditary pancreatitis. The occurrence of pancreatitis in a hereditary way was mainly because of loss of protective mechanisms but the exact cause is not well understood.

PATHOPHYSIOLOGY :

The pathophysiology of acute pancreatitis was not clearly understood. According to previous studies, in the acinar cells of the pancreas there is premature pancreatic enzymes activation which leads to injury of the acinar cells[30]. This activation of the enzymes is mainly because of generation of calcium signals. Activated trypsin mainly leads to the cell injury and it produces cytokines of wihich TNF alpha is the major cytokine which causes pancreatitis[42]. It leads to inflammation in and around the pancreas. There is pancreatic ischemia due to the inflammation, or in some cases may cause pancreatitis. There are many protecting mechanism in the humans against pancreatitis such as SPINK which is a trypsin inhibitor and there also occurs autodegradation of trypsin. There are many ways in which pancreas can regenerate after it was destroyes by the enzymes. Melatonin can favour regeneration by increasing the synthesis of DNA[44]. So in the future with new modalities of investigations and managing techniques, the exact pathogenesis of pancreatitis can be studied and treatment option which is specific for the disease can be explained.

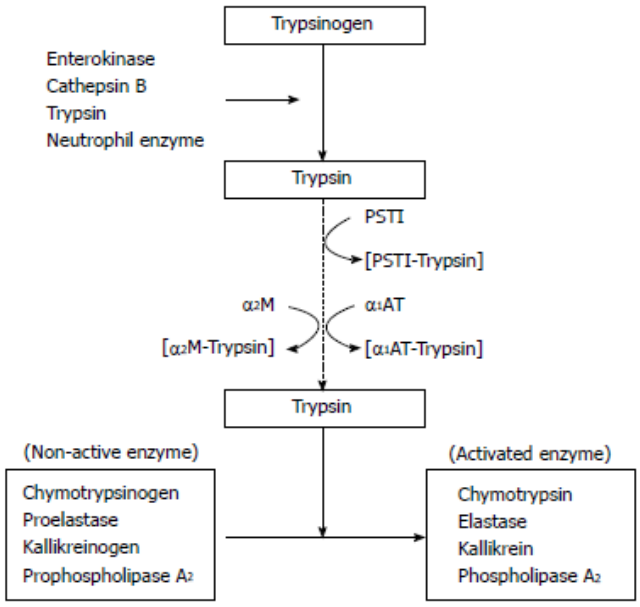


Figure 12 : Suppression mechanism in acute pancreatitis

MAGNITUDE OF THE DISEASE :

The incidence of acute pancreatitis in children is in a rapidly raising trend and there is much increase in the number of admissions with acute or chronic pancreatitis[3,15]. Acute pancreatitis in children is a costly and increasingly recognized disease. In adults also the disease is on the raising trend and the pediatric incidence is also started increasing. This can be attributed to many reasons , due to increase in the incidence of systemic diseases or better health care facility that many children are referred to tertiary care center. The reasons for this increase are not entirely understood and shall be due to multifactorial causes. As many children are referred to tertiary care centres where many

controlled trials are undertaken, it obviously leads to increase in the incidence of pancreatitis but this does not reflect the real situation. There is a correlation regarding investigating the child with more in number of s. amylase and s. lipase tests suggests that there is more proclivity for diagnosing pancreatitis. So the recent increase in the incidence of pancreatitis was due to combination of many factors like recent changes in the diagnosis and managing these children. Pancreatitis in children has become a major burden in both social and economic ways as they have to spend for attending the outpatient department, for getting admitted, for undergoing investigations and many imaging studies which have become costlier nowadays. As the average duration of hospital stay will be more than 5 days, large amount of money was spent on hospital stay itself. In case of surgical intervention, again huge sum of money should be spent which will be a huge burden on the family in the present economic status. They should also spend considerable amount of money for revisits and if they develop any complications. The family members of the child also have loss of pay for bringing the child to hospital and loss of working time[4,45]. So pancreatitis has a significant economic and health burden than what was thought earlier.

PRESENTATION

In the studies done in children, the most common clinical presentation is of abdominal pain and vomiting[19,27,40,46]. Abdominal pain is present in more than 90% of the children presenting with pancreatitis and vomiting around 50 % of the children. The site of the abdominal pain can vary from a child to child. But many children have epigastric pain and some have diffuse abdominal pain[47,48]. Some children have pain localized to the right side of the abdomen. The next common presenting feature is vomiting which may be bilious or non bilious and around 50% children presents with vomiting[3,16,19,20,24,25]. Other common presenting features are fever, distention of the abdomen, any bleeding manifestations like hemetamesis or melena. The epigastric pain can also radiate to the back. The child with acute pancreatitis can also present to the emergency in shock and it can be the only manifestation at the time of admission. In very young children, irritability was a major complaint . The next common presenting feature is vomiting which may be bilious or non bilious and around 50% children presents with vomiting. Other common presenting features are fever, distention of the abdomen, any bleeding manifestations like hemetamesis or melena[19,27,46]. In some cases there is bluish discolouration in the flanks which is termed as Gray Turner sign. The presence of bluish discolouration or ecchymoses near the umbilicus was termed Cullens sign. toddleesn were compared with children between 3 and 20 years of age.

DIAGNOSIS :

Since the pancreas cannot be sampled histologically, it is usually diagnosed by clinical presentation and the investigations like the biochemical parameters and multiple imaging modalities. Many diagnostic criteria have been adapted for diagnosing acute pancreatitis. It includes the presenting symptom which in most cases is abdominal pain, elevation of biochemical parameters liks serum lipase more than thrice of the upper limit of the normal values and ultrasound or computed tomography findings like bulky pancreas, peripancreatic fluid collection, increased echogenecity of the pancreas[1].

Children presents with abdominal pain which is usually noticed in the epigastric region and they can experience reffered pain to the back. In some children, the pain can be diffuse and even lead to rebound tenderness. Other most common presenting features are abdominal distention, fever, bleeding manifestations like hemetamesis or melena. Many diagnostic criteria have been adapted for diagnosing acute pancreatitis. Most of this criteria include the presenting symptom which in most cases is abdominal pain, elevation of biochemical parameters liks serum lipase more than thrice of the upper limit of the normal values and ultrasound or computed tomography findings like bulky pancreas, peripancreatic fluid collection, increased echogenecity of the pancreas

BIOCHEMICAL PRESENTATION

The most common tests done for pancreatitis are serum amylase and serum lipase. The levels of both are elevated in pancreatitis but serum lipase is more specific for pancreatitis than amylase while serum amylase is more sensitive [3,16,27,40]. Serum lipase is elevated in more than 90% of the children while amylase levels are elevated in around 60-80% of children [3,4,20,24,25]. Other investigations done for pancreatitis are total white blood cell count which will be elevated in around 30-40% cases. There will be neutrophilic leukocytosis or relative neutrophilia. C-reactive protein test was also done which will be elevated in the same number of cases. The levels of both are elevated in pancreatitis but serum lipase is more specific for pancreatitis than amylase while serum amylase is more sensitive. Serum lipase is elevated in more than 90% of the children while amylase levela are elevated in around 60-80% of children[20,24,25].

Serum lipase will be elevated more than 3 fold and it is significant and more than 5 fold increase above the baseline value will also be seen. Blood sugar levels may also raise and it should also be monitored. serum amylase and lipase can also get elevated due to non pancreatic reasons so it should also be taken into count before coming to a conclusion. investigations done for pancreatitis are total white blood cell count which will be elevated in around 30-40% cases. There will be neutrophilic leukocytosis or relative neutrophilia. C-

reactive protein test was also done which will be elevated in the same number of cases. Some other biochemical investigations can also be done which are more specific for diagnosing the etiology of pancreatitis. It is of more helpful when we want to diagnose the etiological factors. There are many newer tests like measurement of serum or urine trypsinogen which will be more specific than s. amylase and s. lipase.

IMAGING

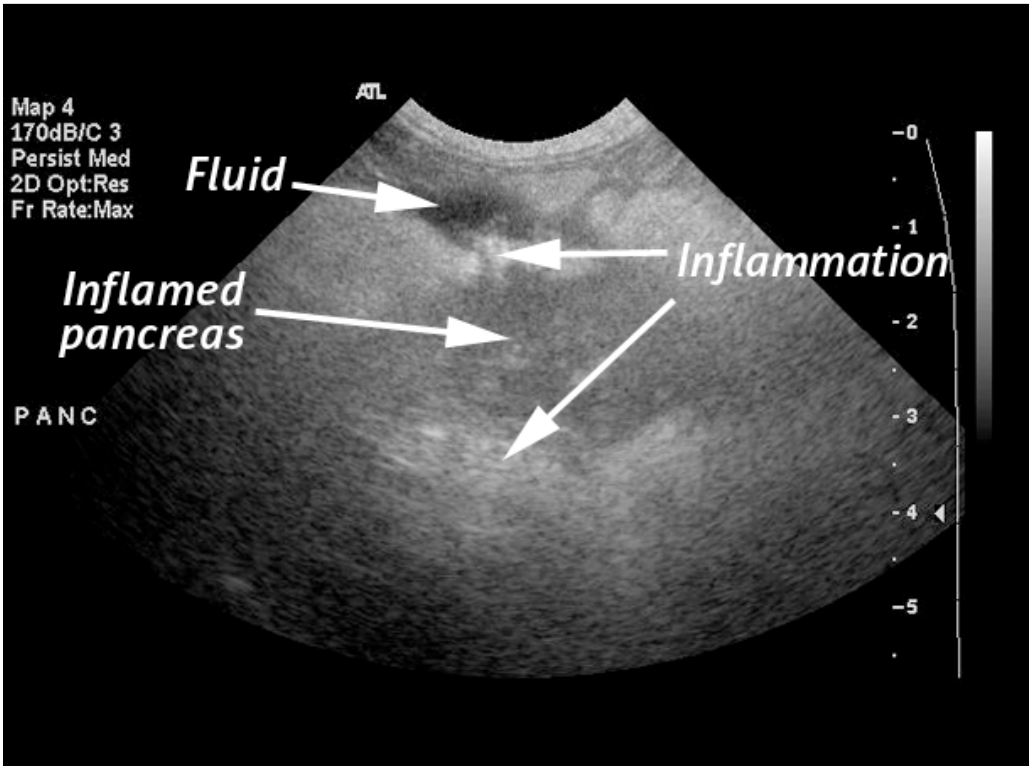


Figure 13 : Ultrasound abdomen showing bulky pancreas and peripancreatic fluid collection

Imaging studies are more helpful in diagnosing pancreatitis. Imaging techniques like ultrasound abdomen and computed tomography scan play a major role in diagnosing pancreatitis. They are done mainly to diagnose pancreatitis but also to detect any other abnormalities in the abdomen like any volvulus, or any intussusceptions. Ultrasound abdomen is very much superior than CT scan because in USG it can detect gall stones in a better way than CT scan. The findings present in USG abdomen are peripancretic fluid collection, bulky pancreas and increased echogenicity in the pancreas [3,16,18,19,27,46,48]. (Figure 13) So it is used primary imaging technique in all children with acute and also in many cases of chronic pancreatitis.

Ultrasonogram of abdomen is very much superior than CT scan because in USG it can detect gall stones in a better way than CT scan[49]. The main demerits in ultrasound technique is that the image cannot be reproduced and there is person to person variability who is doing ultrasound. In most of the children, ultrasound was done in the initial period itself at the time of presentation itself along with other biochemical tests USG was also done and it shows positive findings in a majority of children presenting with acute pancreatitis. Ultrasonogram of abdomen is very much superior than CT scan because in USG it can detect gall stones in a better way than CT scan. The main problems in ultrasound technique is that the image cannot be reproduced and there is person to person variability who is doing ultrasound. Computed

tomogrphy scan is usually not initially recommended as it may not be that much useful in detecting pancreatitis. It is useful after diagnosing pancreatitis to find out any complication like pseudocyst or pancreatic necrosis or calcification [50,51,52]. (Figure 14). There is exposure to radiation and we should be cautious enough to decide whether the person needs the scan by calculating the risk benefit ratio. Other imaging modalities like MRCP and ERCP can also be done during follow up to diagnose the etiology of pancreatitis. Computed tomogrphy scan is usually not initially recommended as it may not be that much useful in detecting pancreatitis. It is useful after diagnosing pancreatitis to find out any complication like pseudocyst or pancreatic necrosis or calcification.

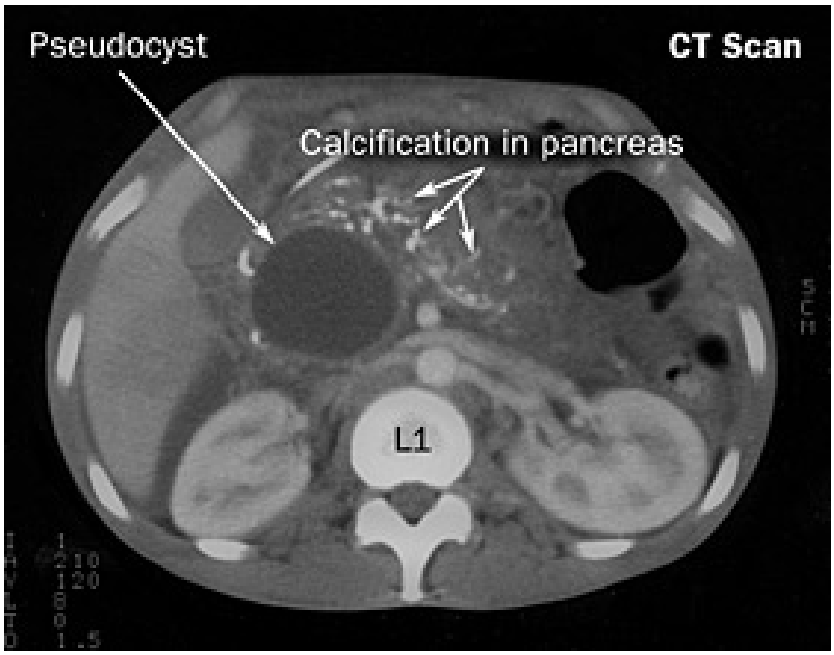


Figure 14 : CT abdomen showing pseudocyst and calcification of pancreas

MRCP & ERCP:

For many biliary and pancreas related condition, nowadays Magnetic resonance cholangopancretography (Figure 15) has largely replaced endoscopic retrgrade cholangopancretography.

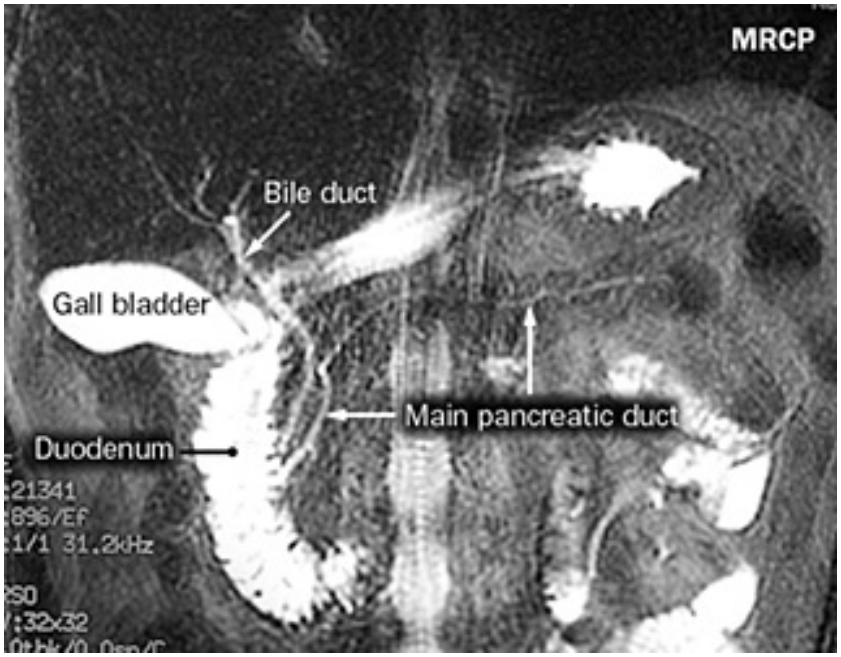


Figure 15 : Magnetic resonance cholangiopancreatography of pancreas

But both of these modalities have same accuracy and both can be of same sensitivity in diagnosing. But with ERCP (Figure 12), endoscopic sphincterotomy and stenting can be done which cannot be done by using Magnetic resonance cholangopancretography.

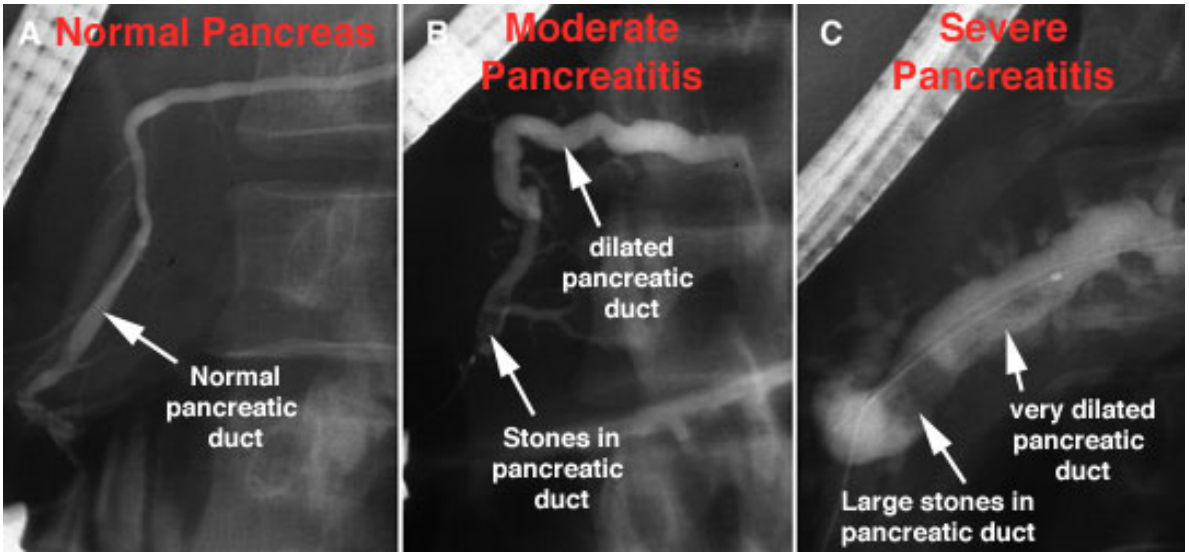


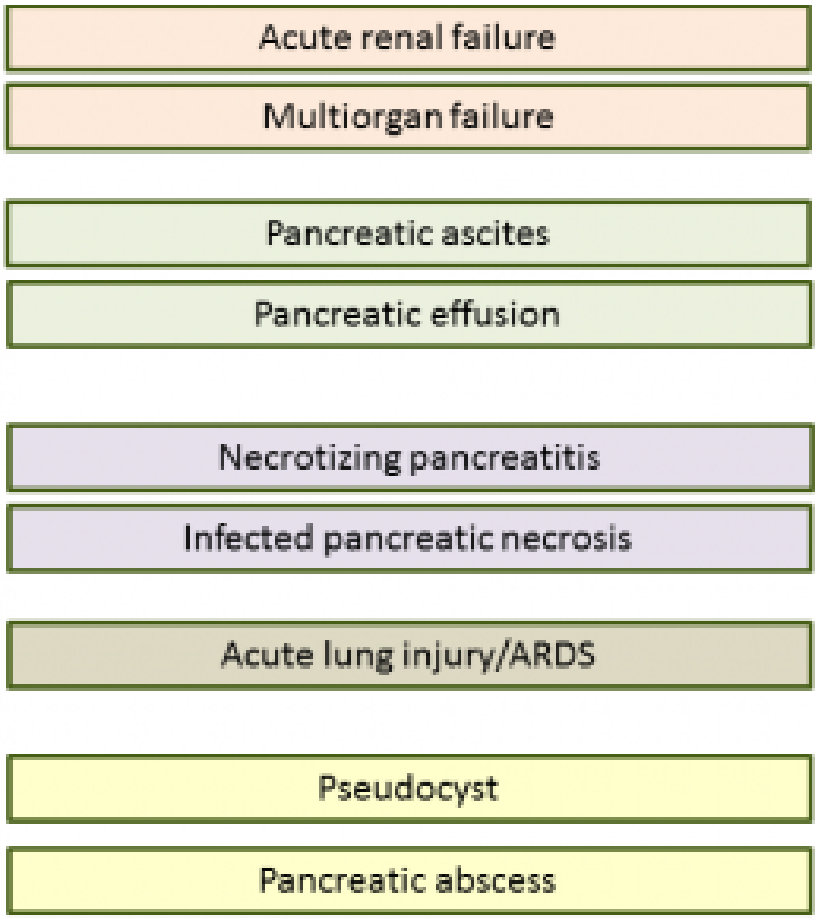
Figure 16 : Endoscopic retrograde cholangiopancreatography of pancreas

TABLE 2: DIFFERENCES BETWEEN MRCP AND ERCP

MRCP	ERCP
Highlight any structure with static fluid	Requires opacification with injected contrast media
Noninvasive so safe esp. in children and pregnant patients	Invasive
Lower cost, faster	20% more expensive than MRCP
No sedation except in few patients	Sedation required
Delineate structures proximal to obstruction.	May fail in patients because of possible tight stricture
No therapeutic intervention	Therapeutic intervention possible
Doesnot use iodine-based com pounds	Requires iodine-based compound usage
Disadvantages	
Duct images obscured by other fluid structures	Risk of pancreatitis
(renal cysts, ascites, pseudocyst)	Intraluminal bleeding
Image artifacts from stents, clips, *etc.*	Duodenal perforation
	Bile leaks
	Stent migration
Contraindications	
Claustrophobic patient	Patient with previous biliary or gastric surgery
Patients with ferromagnetic implants	Patients with high risk profile for general anesthesia

COMPLICATIONS:

The common complications seen in children with pancreatitis are shown in the chart as below.



MANAGEMENT :

Children with acute pancreatitis should be recognised early so that correct treatment measures can be undertaken to reduce the death rate of the patients. The important treatment measures were giving adequate hydration to correct the dehydration of the children. Those children will be suffering from pain, so pain relievers and analgesics should be prescribed. The children suffering from pancreatitis will be poorly nourished because of less oral intake. So they should be given adequate nutrition to meet their health needs. Hypovolemia and decrease in blood pressure can happen due to increase in the permeability of the

endothelium due to inflammatory reponse in pancreatitis children. Thus the intravenous fluids given early in the disease can reduce the ischemia and increase the oxygen delivery to the tissues. So the children presenting with reduced intravascular volume or in shock, usually normal saline fluid should be administered as bolus therapy than to lactated ringer solution. The vital parameter like pulse rate, blood pressure, capillary filling time and pulse volume should be monitored. As the children with pancreatitis will be kept nil by mouth and their oral intake is also poor, maintenance intravenous fluids should be given to maintain their nutrition.

The blood sugar of the patient should be closely monitored as hyperglycemia should always be avoided. As the children with pancreatitis has abdominal pain and nausea, anti diuretic hormone will be secreted which can lead to hyponatremia. As these children have intolerable pain, the analgesics that are used routinely may not be sufficient. Powerful analgesics like tramadol should be given at a dose of 1mg/kg/day two to three doses in a day. Antibiotics and other medications such as octreotide should not be given on a routine basis in all children with pancreatitis.

Initally it was thought that the children with acute pancreatitis should be kept nil by mouth so that it wont stimulate the secretion of pancreatic enzymes and thus it will help in faster recovery of the pancreas. But this causes significant increase in the morbidity of these children. Further, as compared to

enteral nutrition, parenteral nutrition confers more risk of infection to the children. Enteral nutrition which can be given early in cases of pancreatitis may prevent atrophy of the mucosa of the intestine and they maintain their integrity. Further it also plays an important role in prevention of sepsis that occurs due to parenteral nutrition.

There is still confusion regarding the type of enteral nutrition that can be given in all the children with pancreatitis who are suffering from abdominal pain or vomiting, a nasogastric tube should be inserted and it should be kept as drained by gravity so that the bowel can be decompressed . Generally a divergent approach is necessary for treating the children with pancreatitis. If the child develops complication like renal failure, those children should be subjected to hemodialysis and if the children has respiratory difficulty, they should be ventilated mechanically.

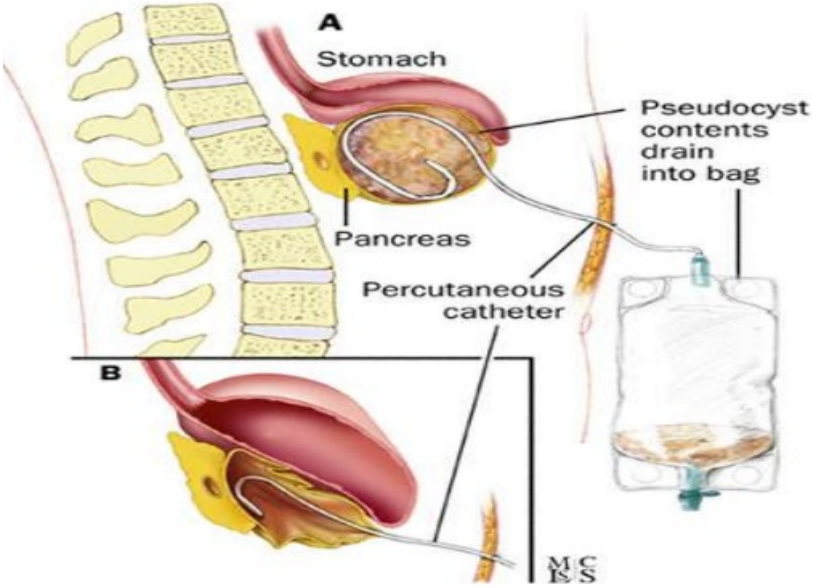


Figure 17: Percutaneous pseudocyst drainage

In case of any fluid collections or pseudocyst, surgical interventions are usually not needed unless they cause compression on other organs. The endoscopic procedures usually performed are endoscopic sphincterotomy with stenting, endoscopic removal of gall stones and drainage of pseudocyst. Pancreatic enzyme supplementation should be given to all children with chronic pancreatitis.

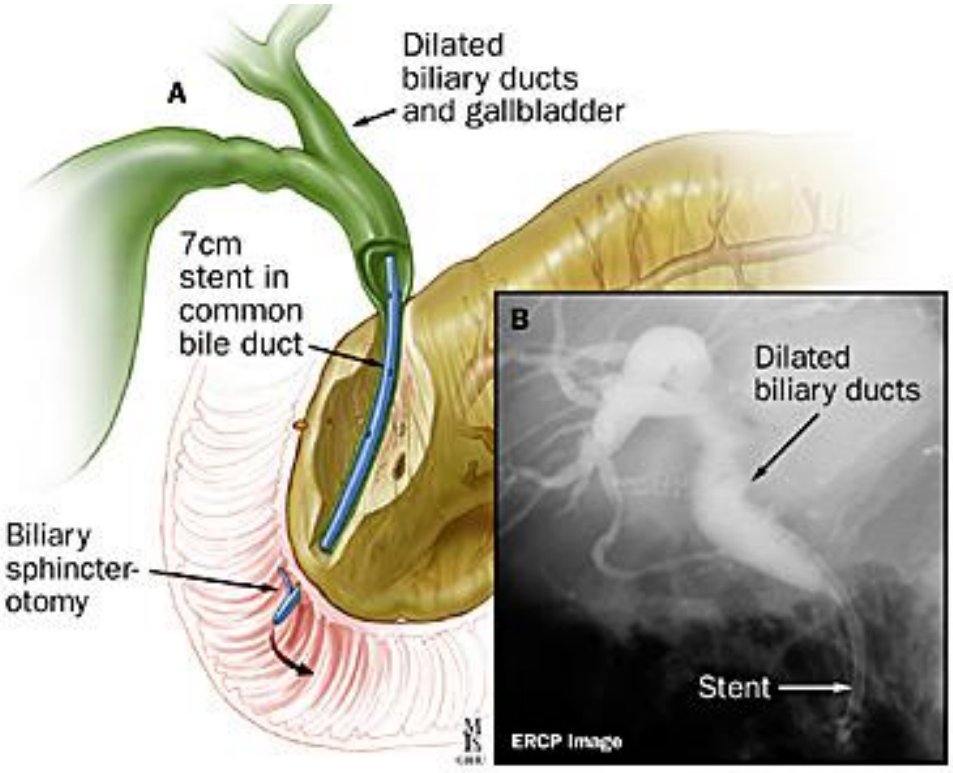


Figure 18: Sphincterotomy with stenting

MATERIALS AND METHODS

Study design:

Descriptive (retrospective and prospective) study

Setting:

PSG hospital, Coimbatore.

Study period: 3 years and 6 months

Retrospective : January 2012 - May 2014

Prospective : June 2014 – July 2015

Sample size :

All 43 children diagnosed to have pancreatitis in PSG hospital during the study period.

INCLUSION CRITERIA :

- All children < 15 years of age in whom a diagnosis of pancreatitis was made between January 2012 and July 2015 .

METHODOLOGY

The study included all children with pancreatitis below the age of 15 years admitted in PSG Hospital from January 2012 to July 2015.

Pancreatitis was suspected clinically if the child presented with severe upper abdominal pain and vomiting with or without fever.

The diagnosis of acute pancreatitis was made if the child met any 2 of the following three criteria 1) classical abdominal pain 2) Elevated serum amylase / serum lipase more than 3 times the upper limit of normal values. 3) radiographic evidence of acute pancreatitis like edema or peripancreatic fluid collection on ultrasound abdomen or CT scan.(3) Pancreatitis was termed as chronic if the abdominal imaging showed pancreatic calcification, duct dilatation or parenchymal atrophy.

The children were managed as per standard protocol, the details of which were left to the discretion of the admitting unit.

For the prospective study, the child was recruited after getting an informed and written consent from the parents. These children were admitted and blood investigations like complete blood counts, C reactive protein, serum amylase, serum lipase, lipid profile and serum calcium and radiological investigations like ultrasound abdomen and xray of the abdomen were done.

History and other details in the proforma were filled up for all the children.

For the retrospective data permission was obtained from hospital authorities for review of case sheets.

CT scan of the abdomen, Magnetic resonance cholangiopancreatography (MRCP), endoscopic retrograde cholangiopancreatography (ERCP) and upper gastrointestinal endoscopy were done in selected children as decided by the treating physician.

RESULTS

The Study population included 43 children of which 28 children (65%) were diagnosed to have acute pancreatitis and 15 children (35%) were diagnosed to have chronic pancreatitis.

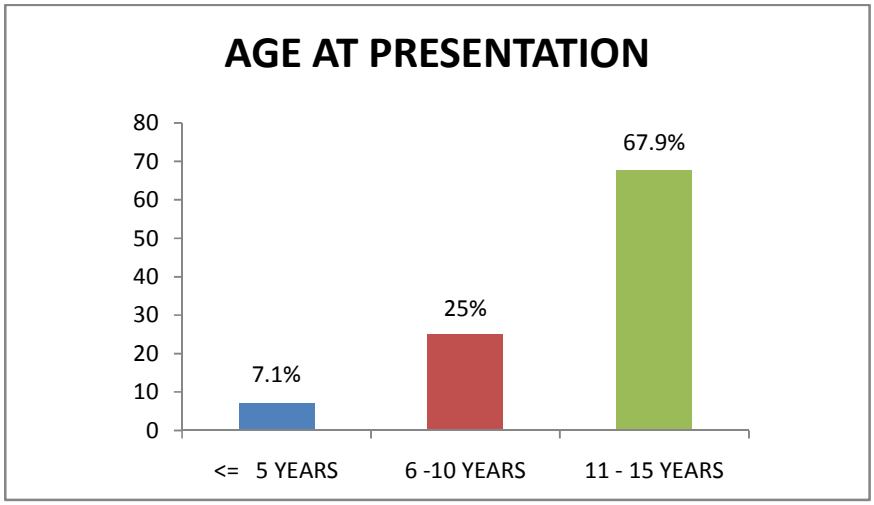
ACUTE PANCREATITIS :

The total number of children diagnosed to have acute pancreatitis were 28.

TABLE 3 : AGE OF THE CHILDREN AT PRESENTATION

AGE	NUMBER OF PATIENTS	PERCENTAGE
≤ 5 YEARS	2	7.1
6 -10 YEARS	7	25
11 - 15 YEARS	19	67.9

FIGURE 19: AGE OF THE CHILDREN AT PRESENTATION

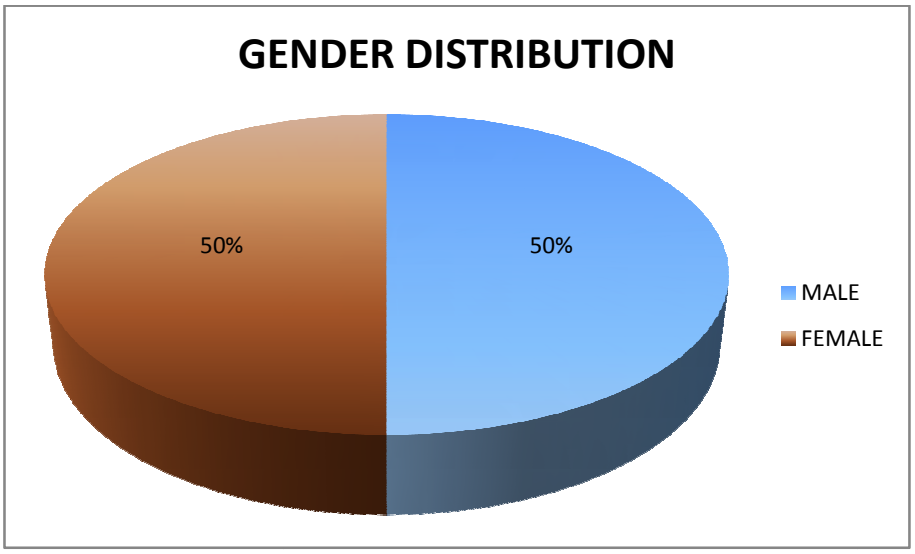


In the study population, among the children with acute pancreatitis, 7.1 % of children were less than 5 years of age; 25% of children were between 6 to 10 years of age and 67.9% of children were between 11 to 15 years of age.(Table 3 and Figure 19).

TABLE 4 : GENDER DISTRIBUTION OF CHILDREN WITH ACUTE PANCREATITIS

GENDER	NUMBER OF PATIENTS	PERCENTAGE
MALE	14	50
FEMALE	14	50

FIGURE 20 : GENDER DISTRIBUTION OF CHILDREN WITH ACUTE PANCREATITIS

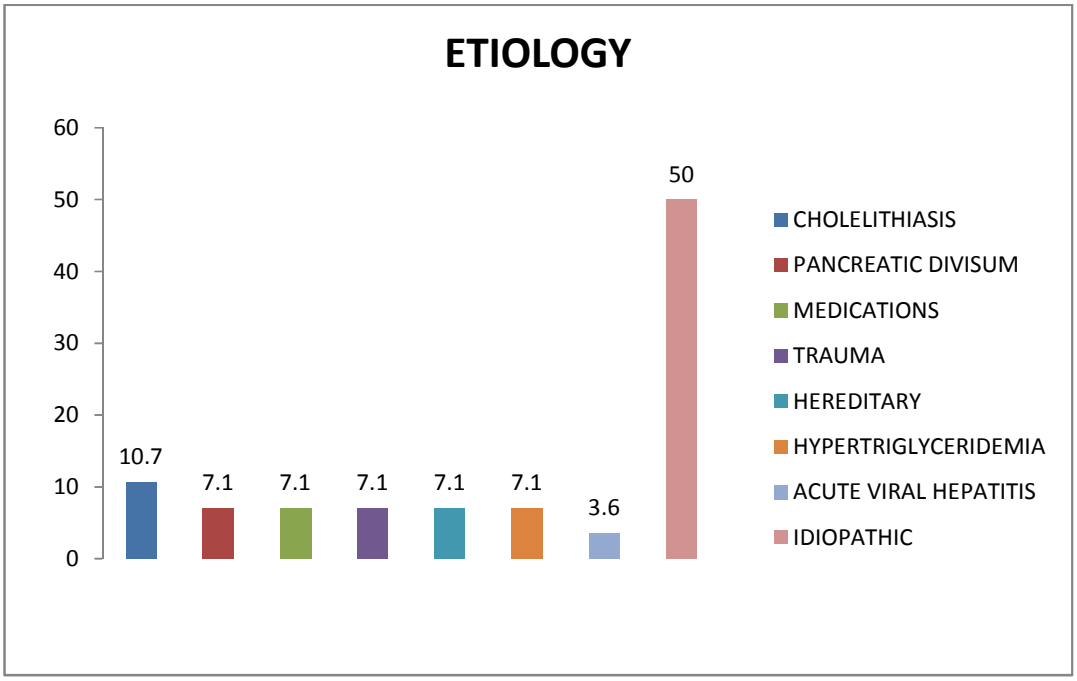


50% of the cases with acute pancreatitis were males (14) and 50 % of the cases were females (14) . Male : female ratio is 1:1. (Table 4 and Figure 20).

TABLE 5 : ETIOLOGICAL FACTORS IN ACUTE PANCREATITIS

ETIOLOGY	**N**	**%**
CHOLELITHIASIS	3	10.7
PANCREATIC DIVISUM	2	7.1
DRUGS(SODIUM VALPROATE)	2	7.1
TRAUMA	2	7.1
HEREDITARY	2	7.1
HYPERTRIGLYCERIDEMIA	2	7.1
ACUTE VIRAL HEPATITIS A	1	3.6
IDIOPATHIC	14	50

FIGURE 21 : ETIOLOGICAL FACTORS IN ACUTE PANCREATITIS

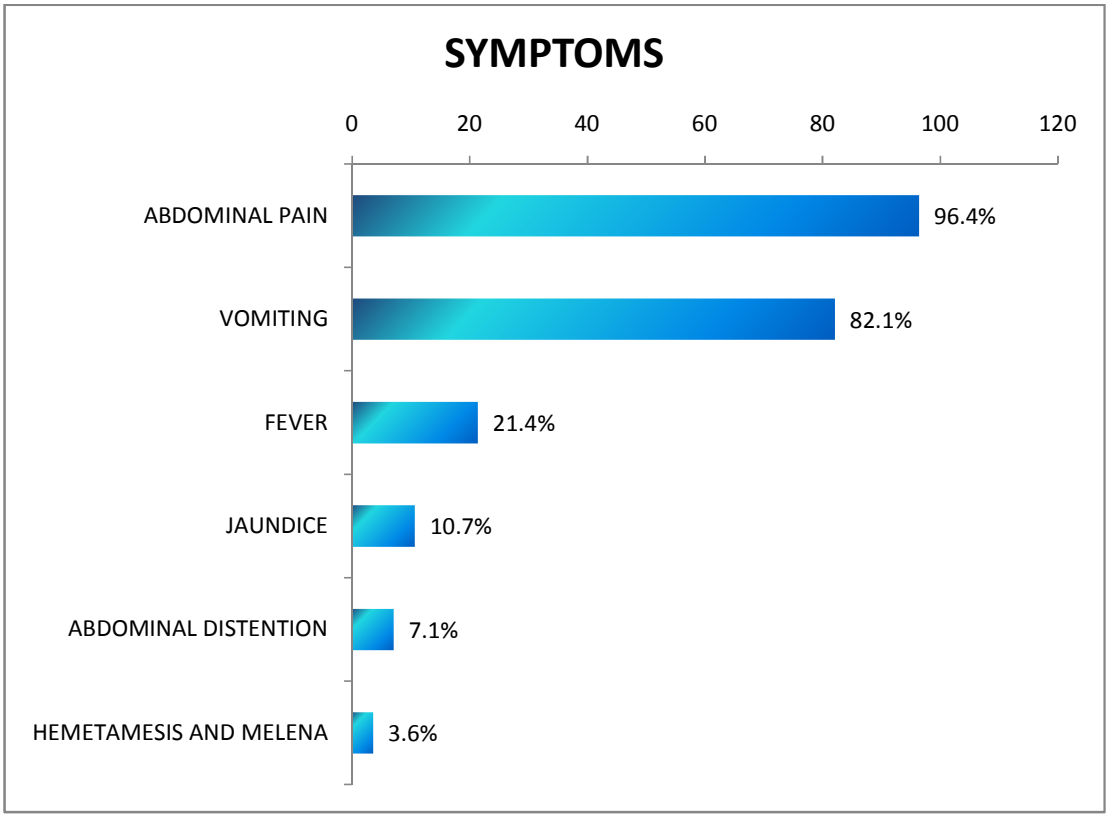


In the study population, among children with acute pancreatitis, about 50 % of children (14) causes were identified of which cholelithiasis constitutes 10.7% ; pancreatic divisum in 7.1% of children; drugs (sodium valproate) in 7.1 % of children ; trauma in 7.1% of children ; hereditary in 7.1% , metabolic cause (hypertriglyceridemia) in 7.1 % of children and infection (acute viral hepatitis) in 3.6 % of children . In the remaining 50 % of children causes were not known and was termed idiopathic. (Table 5 and figure 21).

TABLE 6 : SYMPTOMS IN ACUTE PANCREATITIS

SYMPTOMS	NUMBER	PERCENTAGE
ABDOMINAL PAIN	27	96.4
VOMITING	23	82.1
FEVER	6	21.4
JAUNDICE	3	10.7
ABDOMINAL DISTENTION	2	7.1
HEMETAMESIS AND MELENA	1	3.6

FIGURE 22: SYMPTOMS IN ACUTE PANCREATITIS

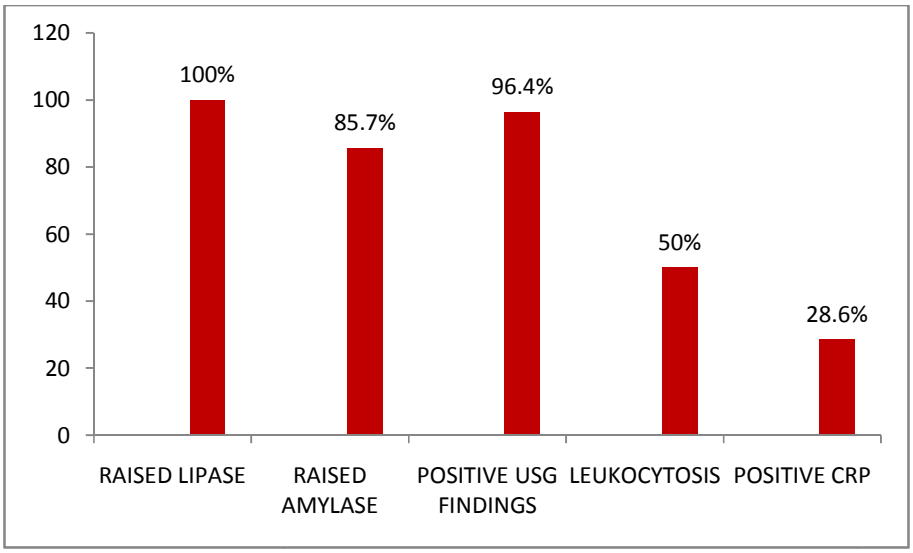


In the study population, among children with acute pancreatitis, 96.4 % of children presented with abdominal pain; 82.1% with vomiting; 21.4 % presented with fever; 10.7 % of children presented with jaundice; 7.1 % with abdominal distention and 3.6% of children presented with hemetamesis/ melena. (Table 6 and figure 22).

TABLE 7 : INVESTIGATIONS IN ACUTE PANCREATITIS

INVESTIGATIONS	N	%
RAISED LIPASE (more than 3 times the normal)	28	100
RAISED AMYLASE (more than 3 times the normal)	24	85.7
POSITIVE USG FINDINGS	27	96.4
LEUKOCYTOSIS	14	50
POSITIVE CRP	8	28.6

FIGURE 23 : INVESTIGATIONS IN ACUTE PANCREATITIS



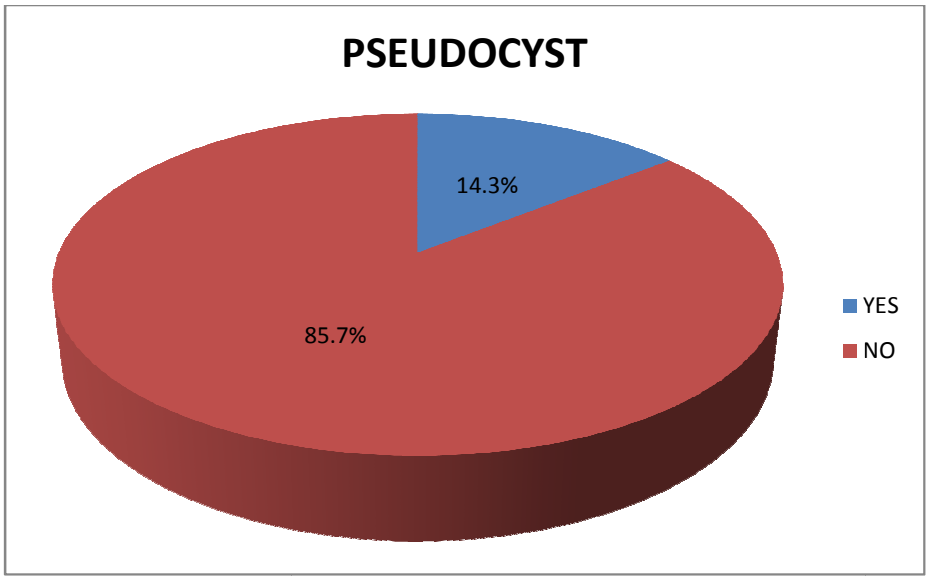
In the study population with acute pancreatitis Serum lipase was elevated in all the 100% of children. Serum Amylase was raised in 85.7 %. Ultrasound abdomen findings suggestive of acute pancreatitis was found in about 96.4% of children. Elevated white blood cells was found in 50% of children; C- reactive protein was positive in 28.6% of children; (Table 7 and Figure 23).

All children (100 %) received intravenous fluids, analgesics and H_2 blockers / proton pump inhibitors. Only 64.3% of children received intravenous antibiotics and 10.7% of children required endoscopic intervention. (endoscopic sphincterotomy with stenting in 1 child; pseudocyst drainage in 1 child and endoscopic removal of gall stones in 1 child).

TABLE 8 : PSEUDOCYST IN ACUTE PANCREATITIS

PSEUDOCYST	N	%
YES	4	14.3
NO	24	85.7

FIGURE 24: PSEUDOCYST IN ACUTE PANCREATITIS

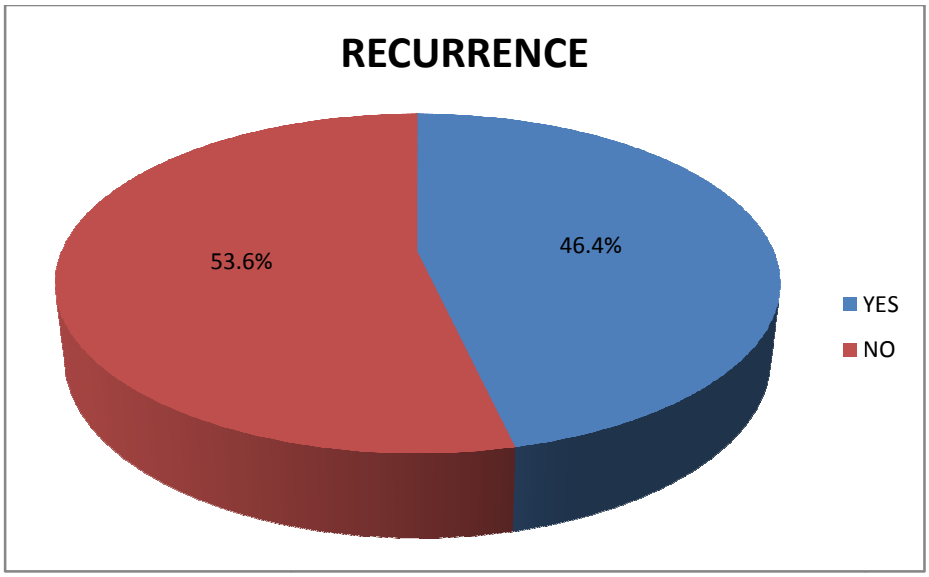


In the study population with acute pancreatitis, only 4 (14.3%) children had developed pseudocyst of pancreas.

TABLE 9 : RECURRENCE RATE IN ACUTE PANCREATITIS

RECURRENCE	N	%
YES	13	46.4
NO	15	53.6

FIGURE 25 : RECURRENCE RATE IN ACUTE PANCREATITIS



In the study population with acute pancreatitis, 46.4% of children had atleast one recurrence of pancreatitis.

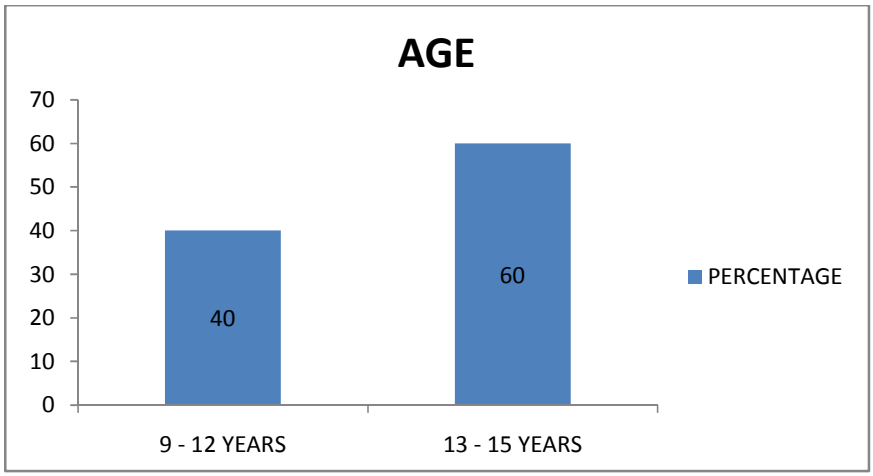
CHRONIC PANCREATITIS

Among the 43 children admitted with pancreatitis, 15 children (35%) were diagnosed as having chronic pancreatitis.

TABLE 10 : AGE DISTRIBUTION OF CHILDREN WITH CHRONIC PANCREATITIS

AGE	NUMBER OF PATIENTS	PERCENTAGE
9 - 12 YEARS	6	40
13 - 15 YEARS	9	60

FIGURE 26 : AGE DISTRIBUTION OF CHILDREN WITH CHRONIC PANCREATITIS

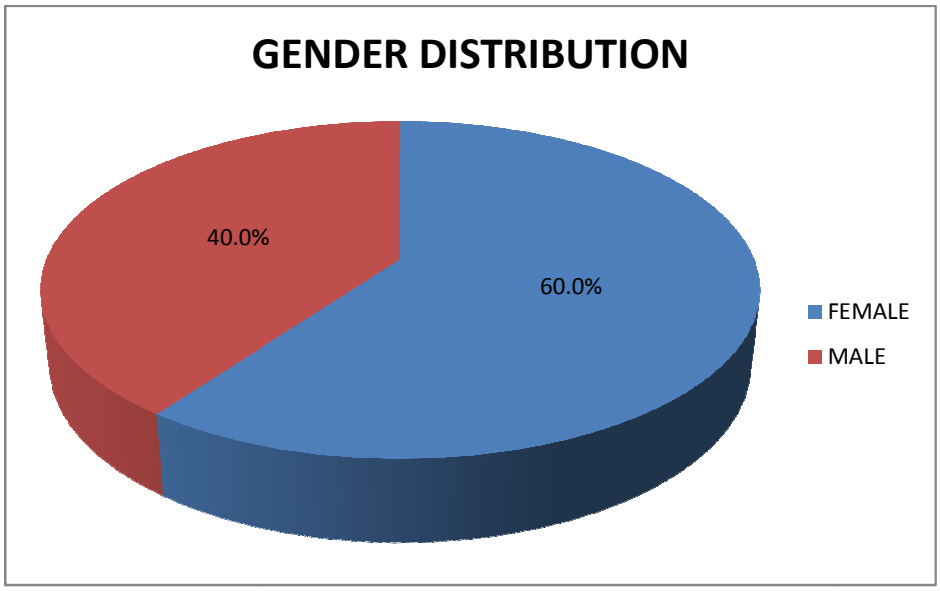


In the study population, among the children with chronic pancreatitis, 40% of the children (6) are in the age group of 9-12 years and 60% of them (9) are between 13-15 years.

TABLE 11 : GENDER DISTRIBUTION IN CHRONIC PANCREATITIS

SEX	NUMBER OF CHILDREN	PERCENTAGE
FEMALE	9	60
MALE	6	40

FIGURE 27 : GENDER DISTRIBUTION IN CHRONIC PANCREATITIS

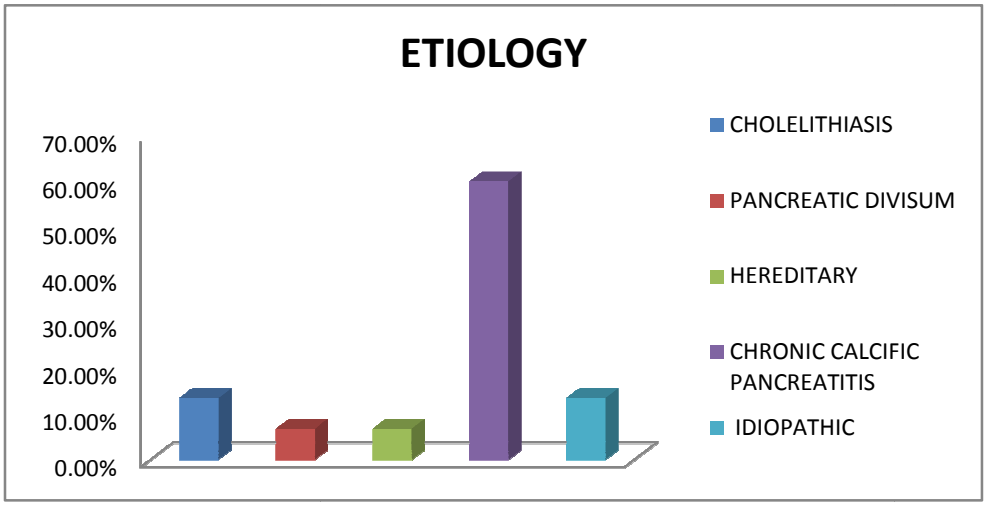


In the study population, among children with chronic pancreatitis, 60% of the children were females and 40% were males

TABLE 12 : ETIOLOGICAL FACTORS IN CHRONIC PANCREATITIS

ETIOLOGY	NUMBER	PERCENTAGE
CHOLELITHIASIS	2	13.3
PANCREATIC DIVISUM	1	6.7
HEREDITARY	1	6.7
CHRONIC CALCIFIC PANCREATITIS	9	60
IDIOPATHIC	2	13.3

FIGURE 28 : ETIOLOGICAL FACTORS IN CHRONIC PANCREATITIS

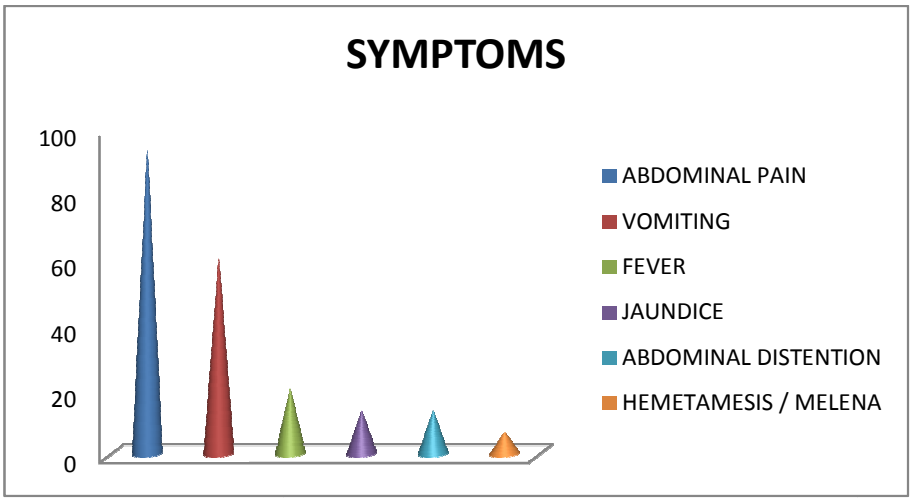


In the study population with chronic pancreatitis, chronic calcific pancreatitis constitutes about 60% of the total cases, cholelithiasis constitutes 13.3 % ; pancreatic divisum 6.7%; and hereditary in 6.7% . In the remaining 13.3% of children causes were not known and was termed idiopathic.

TABLE 13 : SYMPTOMS IN CHRONIC PANCREATITIS

SYMPTOMS	NUMBER	PERCENTAGE
ABDOMINAL PAIN	14	93.3
VOMITING	9	60
FEVER	3	20
JAUNDICE	2	13.3
ABDOMINAL DISTENTION	2	13.3
HEMETAMESIS / MELENA	1	6.7

FIGURE 29 : SYMPTOMS IN CHRONIC PANCREATITIS

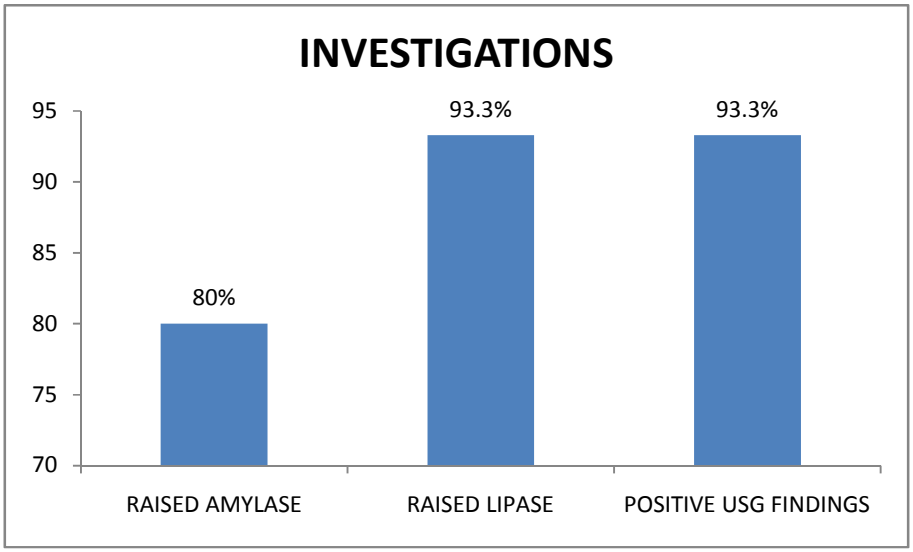


In the study population with chronic pancreatitis, 93.3 % of children presented with abdominal pain; 60 % with vomiting; 20 % presented with fever; 13.3 % of children presented with jaundice; 13.3 % with abdominal distention and 6.7 % of children presented with hemetamesis/ melena.

TABLE 14 : INVESTIGATIONS IN CHRONIC PANCREATITIS

INVESTIGATIONS	N	%
RAISED AMYLASE	12	80
RAISED LIPASE	14	93.3
POSITIVE USG FINDINGS	14	93.3

FIGURE 30 : INVESTIGATIONS IN CHRONIC PANCREATITIS

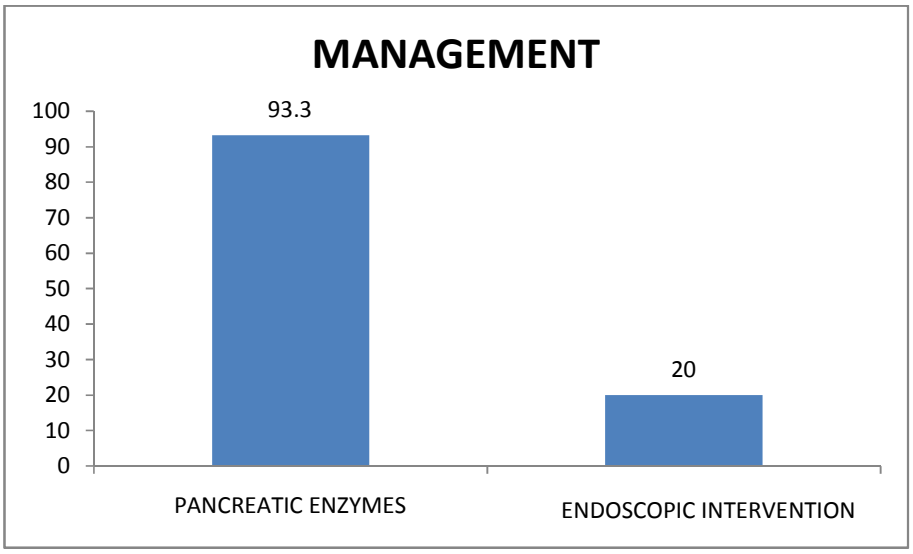


In the study population with chronic pancreatitis, elevated white blood cells was found in 20% of children; C- reactive protein was positive in 20 % of children; Serum Amylase was raised in 80 % while Serum lipase was elevated in 93.3% of children. Ultrasound abdomen findings suggestive of chronic pancreatitis was found in about 93.3% of children.

TABLE 15 : MANAGEMENT IN CHRONIC PANCREATITIS

MANAGEMENT	NUMBER	PERCENTAGE
PANCREATIC ENZYMES	14	93.3
ENDOSCOPIC INTERVENTION	3	20

FIGURE 31 : MANAGEMENT IN CHRONIC PANCREATITIS

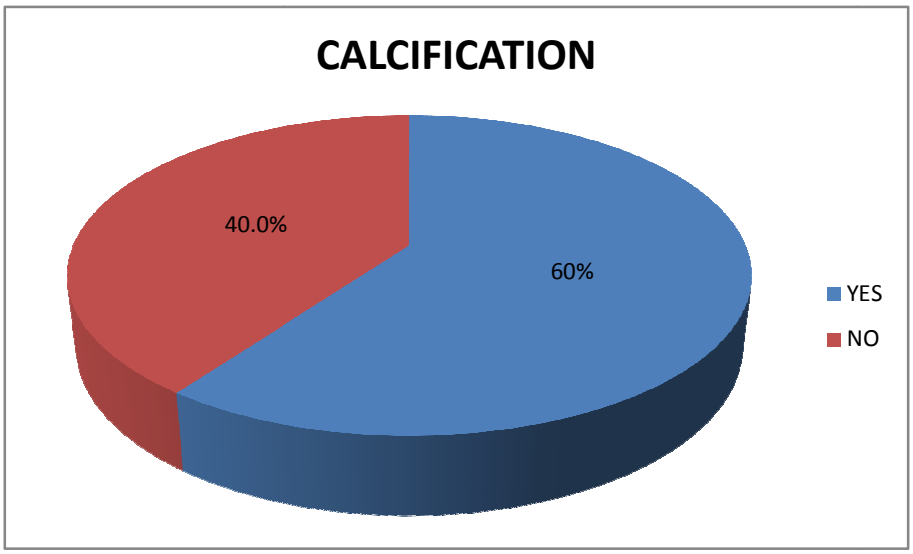


In the study population, among the children with chronic pancreatitis, about 93.3% of children with chronic pancreatitis received pancreatic enzyme supplementation and 20% of children underwent endoscopic intervention. (endoscopic sphincterotomy with stenting in 1 children and endoscopic removal of gall stones in 2 children)

TABLE 16 : CALCIFICATION IN CHRONIC PANCREATITIS

CALCIFICATION	FREQUENCY	PERCENTAGE
YES	9	60
NO	6	40

FIGURE 32 : CALCIFICATION IN CHRONIC PANCREATITIS

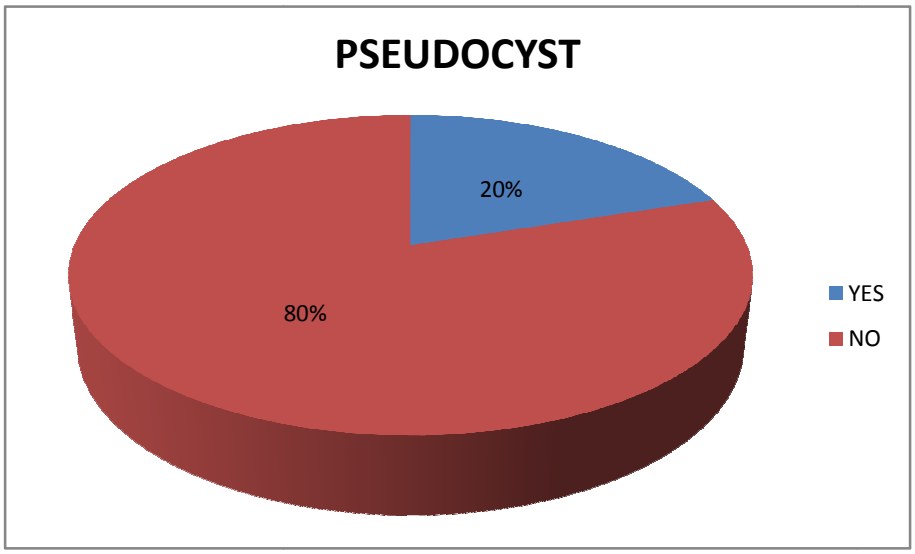


In the study population with chronic pancreatitis, calcification is seen in 60% of children with chronic pancreatitis.

TABLE 17 : PSEUDOCYST IN CHRONIC PANCREATITIS

PSEUDOCYST	FREQUENCY	PERCENTAGE
YES	3	20
NO	12	80

FIGURE 33 : PSEUDOCYST IN CHRONIC PANCREATITIS



In the study population, pseudocyst, as a complication of chronic pancreatitis was found only in 20% of children.

CLINICAL PROFILE AND OUTCOME OF ACUTE PANCREATITIS

PARAMETERS	Number of children	Percentage
SEX (MALE)	14	50
AGE CATEGORIES ≤ 5 YEARS 6 -10 YEARS 11 - 15 YEARS	 2 7 19	 7.1 25 67.9
ABDOMINAL PAIN	27	96.4
VOMITING	23	82.1
FEVER	6	21.4
JAUNDICE	3	10.7
ABDOMINAL DISTENTION	2	7.1
HEMETAMESIS/ MELENA	1	3.6
LEUKOCYTOSIS	14	50
POSITIVE CRP	8	28.6
RAISED AMYLASE	24	85.7
RAISED LIPASE	28	100
POSITIVE USG FINDINGS	27	96.4
ANALGESICS	28	100
IV ANTIBIOTICS	18	64.3
PSEUDOCYST	4	14.3
RECURRENCE	13	46.4
MORTALITY	0	0

CLINICAL PROFILE AND OUTCOME OF CHRONIC PANCREATITIS

PARAMETERS	Number of children	Percentage
SEX (MALE)	6	40
AGE CATEGORIES 9-12 YEARS 13-15 YEARS	 6 9	 40 60
ABDOMINAL PAIN	14	93.3
VOMITING	9	60
FEVER	3	20
JAUNDICE	2	13.3
ABDOMINAL DISTENTION	2	13.3
HEMETAMESIS/ MELENA	1	6.7
RAISED AMYLASE	12	80
RAISED LIPASE	14	93.3
POSITIVE USG FINDINGS	14	93.3
PANCREATIC ENZYMES	14	93.3
PSEUDOCYST	3	20
CALCIFICATION	9	60
MORTALITY	0	0

DISCUSSION

In the present study, among 43 children with pancreatitis, 28 children had acute pancreatitis and 15 children had chronic pancreatitis.

Among the 28 children with acute pancreatitis, 50 % of them were males and 50% were females. This is in accordance with other studies which showed no sex predilection in acute pancreatitis. While in the study by Pezzilli et al it was 50:50; it was 48:52 in the study by Chen & Kong et al and 57:43 in the study by Srikant Das et al in Delhi [25,26,28].

In our study, the mean age at presentation was 11.1 years and the median age was 12.5 years. In the study by Park et al, the mean age at the time of presentation was 13.1 years [4]. In the study by Pezzilli et al, the median age was 10.5 years [26], while in the study by Srikant Das et al it was 10.4 years [28]. In the present study, 7.1% children were less than 5 years of age, 25% were between 5 to 10 years of age and the remaining 67.9% were between 10-15 years of age. This is similar to the observation in the Delhi study where 17.9% were below 5 years of age and 28.6% were between 5 to 10 years of age [28].

In our study, an etiology could be identified in only 50 % of children. Among them, cholelithiasis constituted 10.7%; drugs, hypertriglyceridemia, trauma and hereditary causes constituted 7.1% each and infection (acute viral hepatitis A) in 3.6% of children. In the study from Delhi, etiology could be identified in 65% children. Among them, cholelithiasis constituted 10.7%; drugs, trauma and infections in 14.3% each; hypertriglyceridemia and hereditary causes in 3.6% each[28]. Another study conducted by Werlin et al, cholelithiasis and drugs constituted 12%; trauma and systemic disease in 14% and infections in 8% children [3]. In another study done in Melbourne, etiology could be identified in 74.9% children of which trauma constituted 36.3%, metabolic causes in 5.8%; biliary diseases in 5.4%; drugs in 3.2% and infectious causes in 2.2% [17]. In the study by Chen & Kong et al, etiology was identified in 73.3% children. Among them, systemic diseases were in 22.7%; biliary tract disease constituted 21.3% and trauma in 16% children [25].

In our study, 14 of the 28 children (50%), a cause was not able to be identified. This concurs with the study from Delhi where in 35% of the children etiology could not be identified [28]. In the study from Melbourne, a cause was not identified only in 25% of children [17]. This may be due to very high percentage of trauma in that study.

Abdominal pain was the most common presenting symptom in our study which was seen in 96.4% of children. This data was in accordance with other

studies which reported 95% and above [28,24,25,27]. In our study, vomiting and fever were present in 82.1% and 21.4% of the children respectively. This was in accordance with the Delhi study in which 82% and 33.4% had vomiting and fever respectively. [28]. Another study by Chen & Kong in which vomiting and fever constituted 64.2% and 33.3% respectively[25]. In the study from Taiwan, vomiting and fever were seen in 35.9% and 29.5% children respectively. [24]. This emphasizes the already known fact that fever is not a significant feature of acute pancreatitis in children.

In our study, among the children with acute pancreatitis, jaundice was noted in 10.7% children. This data is comparable with the study done by Srikant Das et al in which jaundice was noted in 15% of the children[28]. Another study by Yeung & Lee et al stated that jaundice was observed in 16.2% of the children with acute pancreatitis[27]. Hemetamesis / melena were present in about 3.6% of children in our study population. This data is similar to the study by Srikant Das et al in which hemetamesis/ melena were observed in 3.9% of children with acute pancreatitis[28].

In our study, serum lipase level was elevated in all the children (100%) while serum amylase was elevated in only 85.7% of the children. This is in accordance with the study by Tiao & Chuang et al in which serum lipase and serum amylase were elevated in 90% and 83.6% of the children respectively [24].Another study by Werlin et al showed elevation of serum lipase and amylase in 83% and 82% of the children respectively [3]. This implies that the diagnosis of acute pancreatitis will be missed in around 15% of children if only serum amylaseis done as the biochemical test.

Ultrasound abdomen findings suggestive of acute pancreatitis was present in 96.4% of the children in our study. This was comparable with Srikant Das et al in which 83.3% of children had positive ultrasound findings[28]. But a study by Tiao & Chuang et al stated that only 78.4% of the children with acute pancreatitis had positive ultrasound findings[24]. Pseudocyst of pancreas was present in 14.3% of children with acute pancreatitis in our study. Other studies had reported 24% and 8%. [25,46]

In the present study, only 10.7% of children required endoscopic intervention (endoscopic sphincterotomy with stenting in 1 child; pseudocyst drainage in 1 child and endoscopic removal of gall stones in 1 child) and the remaining 89.3% children were managed conservatively. Endoscopic intervention was required in 24.5% and 26.7% of children in both the studies

fron China. [24,25]. This is probably due to the higher incidence of biliary tract disease in those studies.

In the present study, in 28 children with acute pancreatitis, 13 (46.4%) of children had recurrence of pancreatitis during the study period. The recurrence rate with other studies were 35.7% and 28%.[26,28] In our study, recurrence was more common in children above 10 years of age. Among, children over 10 years of age, recurrence of acute pancreatitis was seen in 58% compared to 22% of children less than 10 years of age. (P value- 0.13).

In the present study involving 15 children with chronic pancreatitis, mean age at the time of presentation was 12.7 years . In the study by Choudhury et al from Vellore, the mean age at the time of presentation was 15.3 years[31]. In our study, 40% were males and 60% were females, while in the study from vellore, 60% were males and 40% were females[31].

In the present study, etiology could be identified in 86.7% children with chronic pancreatitis. Among them, chronic calcific pancreatitis was seen in about 60% of the children. This is comparable to the study from vellore where it was reported as 68.6% and from Delhi, where it was 65.2%.[28,31]

In our study, cholelithiasis constituted 13.3%, pancreatic divisum in 6.7% and hereditary causes in 6.7% children. In the study from Delhi, cholelithiasis was observed in 4.3%, pancreatic divisum in 4.3% and hereditary causes in 8.6% children.[28]. In the study from Vellore, pancreatic divisum was seen in 8.4% and hereditary cause in 3.1% [31]

In the present study, abdominal pain was the most common presenting symptom in 93.3% of children, while vomiting was present in 60% and fever in 20%. This is similar to the observation of two other studies in which all children had abdominal pain, while fever was seen in only 26% of children.[28,31] In this study, jaundice was noted in 13.3 % of children. This is in accordance with the data by Srikant Das et al from AIIMS, NewDelhi that only 13% of the children with chronic pancreatitis had jaundice[28].

In our study, Serum lipase and amylase were elevated in 93.3% and 80% of children respectively. In another study from Delhi, serum lipase and amylase were elevated in 87.5% and 52.6% children respectively.[28].

Ultrasound abdomen findings suggestive of chronic pancreatitis was found in about 93.3% of children in our study. This is in accordance with the study of Srikant Das et al, in which ultrasound abdomen findings were positive in 95.2% of children[28]. According to Choudhury et al, imaging studied

showed changes consistent with chronic pancreatitis in 100% of children [31]. In the present study, pseudocyst of pancreas was present in 20% of children with chronic pancreatitis. In other studies,pseudocyst was present in 25% and 17.1% of children with chronic pancreatitis. [28,31]

14 of the 15 children with chronic pancreatitis in our study received enzyme supplements. The remaining child was started on enzyme supplements after the study period. All the 15 children were asymptomatic on enzyme supplements and diet modification. In the study by Choudhury et al, all 100% of children received enzyme supplements[31].

Our study confirms that in acute pancreatitis, abdominal pain and vomiting are the most common presenting symptoms, while fever is present in only 21.4% children. Among the investigations, serum lipase and ultrasound abdomen are diagnostic in almost all the children while serum amylase is raised in only 85.7% . The etiology was identified in 50% of children and among them, biliary calculi was the most common cause. Almost 50% of the children had recurrence.

In chronic pancreatitis,the most common cause is chronic calcific pancreatitis. Pseudocyst was seen in 20% of the children. All children were receiving enzyme supplements and remained asymptomatic.

LIMITATIONS:

1. Small number of patients in the study. Pancreatitis in children being unusual, it was difficult to get more number of cases during the study period.

2. MRI Cholangiogram of Genetic mutational analysis were not done for all the children due to financial reasons.

CONCLUSIONS

1) Among the 43 children included in the study, 28 (65%) had acute pancreatitis and 15 (35%) had chronic pancreatitis.

2) The most common identifiable cause of acute pancreatitis was biliary calculi (10.7%) followed by drugs and trauma(7.1%). Around 50% had no identifiable cause.

3) Among the children with acute pancreatitis, more than 95% of the children had upper abdominal pain, and vomiting was present in around 80% of the children while only 20% had fever.

4) All the children with acute pancreatitis had elevated serum lipase while serum amylase was elevated only in 85% of the children.

5) About 46.4% of children who presented with acute pancreatitis had recurrence during the study period. Recurrence was more common in children above 10 years of age. Among the children over 10 years of age, recurrence of acute pancreatitis was seen in 58% compared to 22% of children less than 10 years of age.

6) Among the children with chronic pancreatitis, more than 90% had upper abdominal pain, while vomiting was present in around 60% and only 20% had fever.

7) 93% of the children with chronic pancreatitis had elevated serum lipase levels and 80% had elevated serum amylase levels.

8) The most common identifiable cause of chronic pancreatitis was chronic calcific pancreatitis (60%) followed by cholelithiasis (13.3%) and pancreatic divisum (6.7%). No cause was identified in 13.3% of children.

9) All children with chronic pancreatitis were receiving enzyme supplements and remained asymptomatic.

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ABBREVATIONS

USG	-	Ultrasonogram
CT	-	Computed Tomography
MRCP	-	Magnetic Resonance Cholangio Pancreatography
ERCP	-	Endoscopic Retrograde Cholangio Pancreatography
SGPT	-	Serum Glutamate Pyruvate Transaminase
SGOT	-	Serum glutamic oxaloacetic transaminase
CRP	-	C - reactive protein
SMA	-	Superior mesenteric artery
IVC	-	Inferior vena cava
HUS	-	Hemolytic Uremic Syndrome
SLE	-	Systemic lupus erythematosus
ARDS	-	Acute respiratory distress syndrome
SPINK1	-	Serine protease inhibitor Kazal type 1
PRSS 1	-	protease serine 1
TNF alpha	-	Tumour necrosis factor alpha
DNA	-	Deoxyribonucleic acid

PROFORMA

TITLE OF THE STUDY : Clinical profile and outcome of pancreatitis in children aged less than 15 years.

NAME :

AGE :

SEX :

OP NO. :

IP NO :

HISTORY OF PRESENT ILLNESS (TICK)

a)Abdominal pain ( )
b)Nausea and vomiting ( )
c) hematemesis and melena ( )
d) Abdominal distention ( )
e) Fever (
f)Jaundice ( )

PAST HISTORY (TICK)

a) H/o Recent infection ( )
b) H/o Trauma ( )
c) H/o Drug intake ( )
d) H/o systemic disease ( )
e) Family h/o ( )
f) H/o surgery ( )
g) H/o metabolic disorders ( )

INVESTIGATIONS :

a) complete blood count
b) random blood sugar
c) CRP
d) s.amylase and lipase
e) s.calcium
f) lipid profile
g) liver function test
h) renal function test
i) blood c/s
j) x-ray chest and abdomen
k) USG abdomen
l) CT abdomen
m) MRCP
n) Sweat chloride test
o) Alpha-1 antitrypsin deficiency

TREATMENT :

IV fluids, diet, antibiotics, analgesics, enzyme supplements, surgery

RECURRENCE RATE :

COMPLICATIONS :

SOP 03-V 3.0 / ANX 09-V 2.0

Institutional Human Ethics Committee
PSG Institute of Medical Sciences and Research, Coimbatore

Assent to be in a Research Study
For children between 7-18 years old

Why are we meeting with you?

We want to tell you about something we are doing called a research study. A research study is when doctors collect a lot of information to learn more about something related to health and disease.. Dr Senthil Aakash and some other doctors are doing a study to learn more about CLINICAL PROFILE AND OUTCOME OF PANCREATITIS IN CHILDREN AGED LESS THAN 15 YEARS . After we tell you about it, we will ask if you'd like to be in this study or not.

Why are we doing this study?

We want to find out CLINICAL PROFILE AND OUTCOME OF PANCREATITIS IN CHILDREN AGED LESS THAN 15 YEARS .
So we are getting information from lots of boys and girls like you.
In the whole study, there will be about 45 children.

What will happen to you if you are in this study?

Only if you agree, two things will happen:

1. A small amount of your blood will be drawn. That means it will be taken by a needle in your arm. This will happen only once.

Will this study hurt?

The stick from the needle to draw your blood will hurt, but the hurt will go away after awhile.

Will you get better if you are in this study?
No, this study won't make you feel better or get well. But the doctors might find out something that will help other children like you later.

Will everybody come to know about my condition? (Confidentiality)
We will not tell other people that you are in this research and we won't share information about you to anyone who does not work in the research study

Is this bad or dangerous for me? (Risks involved)
no there is no risk involved

Do I get anything for being in the research?
no

Will you tell me the results?
Yes we will tell u the results when ever you want to know. This results will also be published in a book for research purpose. But we will not reveal your name in it .

Do you have any questions?
You can ask questions any time. You can ask now. You can ask later. You can talk to me or you can talk to someone else.

Do you have to be in this study?
No, you don't. No one will be mad at you if you don't want to do this. If you don't want to be in this study, just tell us. Or if you do want to be in the study, tell us that. And, remember, you can say yes now and change your mind later. It's up to you. *This will not affect in any way your future treatment in this hospital.*

Who can I talk to or ask questions to?
List and give contact information for those people who the child can contact easily (a local person who can actually be contacted). Tell the child that they can also talk to anyone they want to about this (their own doctor, a family friend, a teacher).

If you don't want to be in this study, just tell us. If you want to be in this study, just tell us. This will not affect in any way your future treatment in this hospital.
The doctor will give you a copy of this form to keep.

SIGNATURE OF PERSON CONDUCTING ASSENT DISCUSSION
I have explained the study to _____(*print name of child here*) in language he/she can understand, and the child has agreed to be in the study.

_____ _____
Signature of Person Conducting Assent Discussion Date

_____
Name of Person Conducting Assent Discussion (*print*)

Dr. SENTHIL AAKASH.K

Certificate of Assent

I have read this information (or had the information read to me) I have had my questions answered and know that I can ask questions later if I have them.

I agree to take part in the research.

OR

I do not wish to take part in the research and I have not signed the assent below._____ (initialed by child/minor)

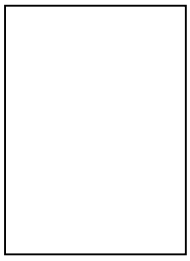
Only if child assents:
Print name of child _____
Signature of child: _____
Date:_____
day/month/year

If illiterate:

A literate witness must sign (if possible, this person should be selected by the participant, not be a parent, and should have no connection to the research team). Participants who are illiterate should include their thumb print as well.

I have witnessed the accurate reading of the assent form to the child, and the individual has had the opportunity to ask questions. I confirm that the individual has given consent freely.

*Print name of witness (not a parent)*_____ *AND* *Thumb print of participant*
Signature of witness _____
Date _____
Day/month/year



I have accurately read or witnessed the accurate reading of the assent form to the potential participant, and the individual has had the opportunity to ask questions. I confirm that the individual has given assent freely.

Print name of researcher_____

PSG Institute of Medical Science and Research, Coimbatore
Institutional Human Ethics Committee
INFORMED CONSENT FORMAT FOR RESEARCH PROJECTS

I K. SENTHIL AAKASH carrying out a study on the topic: **CLINICAL PROFILE AND OUTCOME OF PANCREATITIS IN CHILDREN AGED LESS THAN 15 YEARS – DESCRIPTIVE(RETROSPECTIVE AND PROSPECTIVE) STUDY**

My research guide is: Dr.JOHN MATTHAI

The justification for this study is there is a paucity of literature regarding the profile of pancreatic disorders in children. The divergence of opinion between authors regarding etiology, pathophysiology, definitions and classification of pancreatic disorders and non-uniformity of vocabulary makes it difficult to compare different studies. From our country, only case reports of various pancreatic disorders in children were available. The present study is to determine the profile and outcome of pancreatic disorders in children presenting to our hospital

The objectives of this study are:

Primary Objective: To study the etiology and presentation of acute and chronic pancreatitis in children and to manage accordingly

Secondary Objective: To determine the complication and recurrence rate of both acute and chronic pancreatitis in children.

Sample size: 45

Study volunteers / participants are (specify population group & age group)

Children with pancreatitis (less than 15 years old)

Location: Department of paediatrics, PSG IMSR

We request you to kindly cooperate with us in this study. We propose collect background information and other relevant details related to this study. We will be carrying out:

.

Data collected will be stored for a period of five years We will not use the data as part of another study.

Clinical examination AND
Blood sample collection: it varies depending upon the clinical condition of the child .

Whether blood sample collection is part of routine procedure or for research (study) purpose: routine procedure

Specify **purpose**, discomfort likely to be felt and side effects, if any: no discomfort or side effects

Whether blood sample collected will be stored after study period: No, it will be destroyed

Whether blood sample collected will be sold: No

Whether blood sample collected will be shared with persons from another institution: No

Medication given, if any, duration, side effects, purpose, benefits: no medication is used.
Benefits from this study : identification of most common etiological factors, presenting symptoms, complications involved and rate of recurrence in pancreatitis patients.

Risks involved by participating in this study :no risks involved

How the **results** will be used: results will be used to assess the morbidity pattern of pancreatitis patients .

If you are uncomfortable in answering any of our questions during the course of the interview / biological sample collection, **you have the right to withdraw the baby from the study at anytime.** You have the freedom to withdraw your child from the study at any point of time. Kindly be assured that your refusal to participate or withdrawal at any stage, if you so decide, will not result in any form of compromise or discrimination in the services offered nor would it attract any penalty. You will continue to have access to the regular services offered to your child. You will **NOT** be paid any remuneration for the time you spend with us for this interview / study. The information provided by you will be kept in strict confidence. Under no circumstances shall we reveal the identity of the respondent or their families to anyone. The information that we collect shall be used for approved research purposes only. You will be informed about any significant new findings - including adverse events, if any, developed during the course of this research which may relate to your willingness to continue participation.

Consent: The above information regarding the study, has been read by me/ read to me, and has been explained to me by the investigator/s. Having understood the same, I hereby give my consent to them to enroll my child in this study. . I am affixing my signature / left thumb impression to indicate my consent and willingness to enroll my baby in this study (i.e., willingly abide by the project requirements).

Signature / Left thumb impression of the parent :

Signature of the researcher:
Contact no:9600939977 . department extension number : 5140 ,
tks.cbe@gmail.com
IHEC CONTACT NO:0422 2570170 EXTENSION NO:5818
Witness:

Institutional Human Ethics Committee
PSG Institute of Medical Sciences and Research, Coimbatore

Parental Consent Form

Title of Study: CLINICAL PROFILE AND OUTCOME OF PANCREATITIS IN CHILDREN AGED LESS THAN 15 YEARS – DESCRIPTIVE(RETROSPECTIVE AND PROSPECTIVE) STUDY

Name of the Principal Investigator: K. SENTHIL AAKASH

Department: PAEDIATRICS

Your child is invited to participate in a study of pancreatitis in children based on their presentation and outcome.

My name is **K. SENTHIL AAKASH** and I am a Post Graduate at PSG Institute of Medical Sciences and Research, Coimbatore. This study is done to know the manifestations and outcome of pancreatitis in children So that it would be an evidence for management of the condition in future.

I am asking for permission to include your child in this study because I expect to have **Minimum 45** participants in the study.

If you allow your child to participate, I will collect medical details from your babies case record.

Any information that is obtained in connection with this study and that can be identified with your child will remain confidential and will be disclosed only with your permission. His or her responses will not be linked to his or her name or your name in any written or verbal report of this research project.

Your decision to allow your (son/daughter/child/infant/adolescent youth) to participate will not affect your or his or her present or future relationship with PSGIMS&R or PSG Hospitals. If you have any questions about the study, please ask me. If you have any questions later, call me at 9600939977 If you have any questions or concerns about your (son/daughter/child/infant/adolescent youth)'s participation in this study, call 9600939977.

You may keep a copy of this consent form.
You are making a decision about allowing your child to participate in this study. Your signature below indicates that you have read the information provided above and have decided to allow him or her to participate in the study. If you later decide that you wish to withdraw your permission for your child to participate in the study, simply tell me.

You may discontinue his or her participation at any time. *This will not affect in any way your future treatment in this hospital.*

Printed Name of the child :

Signature of Parent(s) or Legal Guardian with Date Signature of Investigator with Date

மனித உரிமை கோட்பாடுகள் குழு

PSG மருத்துவக் கல்லூரி மற்றும் மருத்துவமனை, கோவை

இந்த ஆராய்ச்சி பதினைந்து வயதுக்குட்பட்ட குழந்தைகளிடம் செய்யப்படுகிறது.

நாம் எதற்கு சந்திக்கிறோம் ?

நாங்கள் அராய்ச்சி படிப்பைப் பற்றிக் கூற விரும்புகிறோம். ஆராய்ச்சி படிப்பு என்பது மருத்துவர்கள் ஒரு குறிப்பிட்ட மருத்துவ தகவல்களை சேகரித்து அதைப் பற்றி தெரிந்து கொள்வது.

நான் Dr. க. செந்தில் ஆகாஷ், பதினைந்து வயதுக்குட்பட்ட குழந்தைகளில் கணைய அழற்சி (பேன்கிரியாடைட்டிஸ்) நோயின் தன்மை மற்றும் விளைவுகளை ஆராய்கிறேன்.

எதற்காக இந்தப் பரிசோதனை ?

இதன் மூலம், பதினைந்து வயதுக்குட்பட்ட குழந்தைகளில் கணைய அழற்சி (பேன்கிரியாடைட்டிஸ்) நோயின் தன்மை மற்றும் விளைவுகளை தெரிந்து கொள்வதற்காக, இதற்காக நிறைய தகவல்களை ஆண் மற்றும் பெண் குழந்தைகளிடம் இருந்து பெறுகிறோம்.

இந்த ஆய்வில் மொத்தமாக 45 குழந்தைகள் உள்ளனர். இந்த பரிசோதனை மூலம் உங்களுடைய தொந்தரவுகள் சரிசெய்யப்படமாட்டாது.

ஆனால் இந்த பரிசோதனை மூலம் பிற குழந்தைகளுக்கு உபயோகமாக இருக்கும்.

உங்களின் பரிசோதனையின் முடிவுகள் வேறு யாருக்கும் செய்யப்பட மாட்டாது.

உங்களுக்கு மட்டுமே தெரியப்படுத்தப்படும் இந்த பரிசோதனையில் உங்களுக்கு சந்தேகம் இருந்தால் டாக்டர். செந்தில் ஆகாஷ் – 9600939977 தொடர்புக்கொள்ளவும்.

இந்த பரிசோதனையில் நீங்கள் பங்கு கொள்ள வேண்டும் என்ற கட்டாயம் இல்லை.

உங்களுக்கு விருப்பம் இல்லை என்றால் இதில் இருந்து எப்போதும் வேண்டுமானாலும் விலகிக் கொள்ளலாம்.

இதனால் இந்த மருத்துவமனையின் எந்த சலுகைகளும் குறைக்கப்பட மாட்டாது.

டாக்டர். செந்தில் ஆகாஷ் ஆகிய நான் இந்த சோதனையைப் பற்றி அனைத்து தகவல்களையும், குழந்தைகளின் பெற்றோர்களுக்கு தமிழில் கூறியுள்ளேன்.

குழந்தையின் பெற்றோர் அதை நன்கு புரிந்து பிறகு இதற்கு சம்மதம் தெரிவித்துள்ளனர்.

உங்களிடம் இந்த நோயைப் பற்றி சில கேள்விகள் கேட்கப்படும். புதிய பரிசோதனை எதுவும் செய்யப்படாது.

இந்த பரிசோதனை வழக்கமாக செய்யப்படும் பரிசோதனை மட்டுமே.

இந்த பரிசோதனையின் முடிவை மட்டுமே எனது ஆய்வுக்கு எடுத்துக்கொள்வேன்.

ஆய்வுக்குட்பட்டவரின் கையொப்பம் **ஆய்வாளரின் கையொப்பம்**

தேதி :

பகுதி – 2

சம்மத சான்று

நான் இந்தக் தகவலைப் படித்தேன் (அல்லது) தகவலை படிக்கக் கேட்டேன் எனது கேள்விகளுக்குப் பதில் அளிக்கப்பட்டது மற்றும் கேள்விகள் எதுவும் இருந்தால் அவைகளை வருங்காலத்தில் கேட்கலாம் என்பதும் எனக்குத் தெரியும்.

நான் இந்த ஆராய்ச்சியில் பங்கேற்க சம்மதிக்கிறேன்.

அல்லது

நான் இந்த ஆராய்ச்சியில் பங்கேற்க விருப்பம் இல்லை மற்றும் கீழே சம்மதம் என்று கையொப்பம் செய்யவில்லை. ..... .

(குழந்தை / மைனர் / இனிசியல் செய்யப்பட்டுள்ளது)

குழந்தை மட்டும் சம்மதம் தெரிவித்தால்

குழந்தையின் பெயர் :

குழந்தையின் கையெழுத்து :

தேதி :

படிப்பறிவு இல்லாதவராக இருந்தால் படிப்பறிவு உள்ள சாட்சி கையொப்பம் செய்ய வேண்டும் (முடிந்தால் இந்த நபர் கலந்து கொள்வபரால் தேர்ந்தெடுக்கப்பட வேண்டும். இந்த நபர் பெற்றோராக இருக்கக்கூடாது. மற்றும் அவருக்கு ஆராய்ச்சி குழுவிடம் எந்த தொடர்பும் இருக்கக்கூடாது).

நான் சம்மதம் படிவத்தை குழந்தையிடம் சரியாக படித்துக் காண்பித்ததைப் பார்த்தேன். மற்றும் அந்த நபருக்கு கேள்விகள் கேட்க வாய்ப்பு இருந்தது. அந்த நபர் தனது சம்மதத்தை முழு மனதுடன் கொடுத்தார் என்பதை நான் உறுதி கூறுகிறேன்.

சாட்சிகள் :

சாட்சியின் கையெழுத்து :

தேதி :

கலந்து கொள்பவரின் இடது
பெருவிரல் ரேகை

ஆராய்ச்சி செய்பவரின்

பெயர் : டாக்டர் K. செந்தில் ஆகாஷ்

கைபேசி எண். 96009 39977

MASTER CHART

Acute Pancreatitis:

S.NO	IP NO	OP NO	AGE	SEX	ABDOMINAL PAIN	VOMITING	FEVER	JAUNDICE	ABDOMINAL DISTENTION	HEMATAMESIS AND MELENA	CHOLELITHIASIS	PANCREATIC DIVISUM	DRUG INDUCED	HEREDITARY	TRAUMA	METABOLIC DISORDERS	RECENT INFECTION	LEUKOCYTOSIS	RAISED CRP	RAISED S. AMYLASE	RAISED S. LIPASE	ABNORMAL LIPID PROFILE	ABNORMAL LFT	POSITIVE USG FINDINGS	ABNORMAL XRAY	POSITIVE CT/ MRI FINDINGS	IV FLUIDS	IV ANTIBIOTICS	ANALGESICS	H2 BLOCKERS/ PPIs	ENDOSCOPIC INTERVENTION	DURATION OF HOSPITAL STAY	PSEUDOCYST	RECURRENCE
1	I12003620	O11081959	8	M	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	0	0	1	0	2	1	0	1	1	0	5	0	0
2	I13027678	O11075628	14	F	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	0	0	1	0	0	1	0	1	1	0	4	0	1
3	I14019181	O11075651	12	M	1	0	1	0	0	0	0	0	0	0	0	0	0	1	0	1	1	2	0	1	2	2	1	1	1	1	0	7	1	1
4	I14030531	O14073448	14	F	1	1	0	0	0	0	1	0	0	0	0	0	0	1	0	1	1	0	0	1	2	0	1	1	1	1	0	6	0	1
5	I15021321	O10074650	12	F	1	1	0	0	0	0	0	0	0	1	0	0	0	0	0	1	1	2	2	1	2	2	1	1	1	1	0	5	0	1
6	I12022441	O12056218	9	M	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	1	0	2	1	0	1	1	0	4	0	0
7	I13014394	O13032953	13	M	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	2	2	1	2	2	1	0	1	1	0	3	0	1
8	I14020330	O14048253	11	F	1	1	1	0	0	0	0	0	0	0	0	0	0	1	1	1	1	0	0	1	1	0	1	1	1	1	0	6	0	0
9	I15000216	O15000451	6	F	1	0	1	1	1	0	0	0	0	0	0	0	1	0	0	1	1	0	0	1	0	2	1	0	1	1	0	6	0	0
10	I14023521	O14057031	6	F	1	1	0	0	0	0	1	0	0	0	0	0	0	1	0	1	1	0	1	1	0	0	1	1	1	1	1	8	0	1
11	I12027792	O12070571	14	M	1	1	1	0	0	0	0	0	0	0	0	0	0	1	1	1	1	0	0	1	0	0	1	1	1	1	0	5	1	0
12	I12027650	O12070153	5	M	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	0	0	0	2	2	1	1	1	1	0	6	0	0
13	I12008492	O12020967	13	F	1	0	0	0	0	0	0	0	1	0	0	0	0	1	0	1	1	0	1	1	0	2	1	1	1	1	0	5	0	0
14	I12008376	O12020741	6	F	1	1	0	1	0	0	0	1	0	0	0	1	0	1	1	1	1	0	1	1	2	2	1	1	1	1	0	7	0	0
15	I12028486	O11075628	15	F	1	1	0	0	0	0	0	0	0	0	0	0	0	1	0	1	1	0	0	1	2	2	1	0	1	1	0	3	0	0
16	I12027704	O10074650	12	F	1	1	0	0	0	0	0	0	0	1	0	0	0	0	0	1	1	0	0	1	0	2	1	0	1	1	0	7	0	0
17	I13037959	O13090736	4	F	0	1	0	0	0	1	0	0	0	0	0	0	0	0	0	1	1	0	0	1	0	2	1	1	1	1	0	7	0	1
18	I13033983	O13065163	15	M	1	1	1	0	0	0	0	0	0	0	0	0	0	0	1	1	1	0	0	1	0	2	1	1	1	1	0	6	0	1
19	I12000551	O12001397	15	M	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	0	0	1	0	1	1	0	1	1	0	5	0	1
20	I12001258	O12003360	15	F	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	0	0	1	0	1	1	0	1	1	0	4	0	1
21	I13000269	O13000566	15	F	1	1	0	0	0	0	0	0	0	0	0	0	0	1	1	1	1	0	0	1	0	1	1	1	1	1	0	6	0	0
22	I13002734	O13005995	14	M	1	1	0	0	0	0	0	0	0	0	1	0	0	0	0	1	1	0	0	1	0	1	1	1	1	1	0	4	0	1
23	I13023861	O13056049	13	F	1	1	0	0	0	0	1	0	0	0	0	0	0	1	1	1	1	0	0	1	1	0	1	1	1	1	1	4	0	1
24	I14004130	O13052326	15	M	1	1	0	1	1	0	0	1	0	0	0	1	0	1	0	0	1	1	1	1	0	1	1	1	1	1	0	4	0	1
25	I15002265	O15004954	15	M	1	1	0	0	0	0	0	0	1	0	0	0	0	1	1	1	1	0	0	1	0	1	1	1	1	1	0	8	1	0
26	I14020448	O14048503	6	M	1	1	1	0	0	0	0	0	0	0	0	0	0	1	0	1	1	0	0	1	0	2	1	1	1	1	0	4	0	0
27	I14014214	O14033707	6	M	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	1	0	1	1	0	1	1	1	4	0	0
28	I14007595	O14019356	9	M	1	1	0	0	0	0	0	0	0	0	1	0	0	1	1	1	1	0	0	1	1	1	1	1	1	1	0	15	1	0

Chronic Pancreatitis:

S.NO	IP NO	OP NO	AGE	SEX	ABDOMINAL PAIN	VOMITING	FEVER	JAUNDICE	ABDOMINAL DISTENTION	HEMATAMESIS AND MELENA	DRUG INDUCED	CHOLELITHIASIS	PANCREATIC DIVISUM	FAMILY HISTORY	LEUKOCYTOSIS	RAISED CRP	RAISED S. AMYLASE	RAISED S. LIPASE	ABNORMAL LIPID	ABNORMAL LFT	ABNORMAL USG ABDOMEN	ABNORMAL CT/MRI	MRCP/ ERCP	GENETIC ANALYSIS	IV FLUIDS	IV ANTIBIOTICS	ANALGESICS	H2 BLOCKERS/ PPIs	DIETARY MODIFICATIONS	PANCREATIC ENZYMES	ENDOSCOPIC INTERVENTIO	PSEUDOCYST	ASCITES	CALCIFICATION
1	I14017568	O14041589	13	F	1	0	0	0	0	0	0	0	0	0	0	0	1	1	0	0	1	1	1	2	1	0	1	1	1	1	0	0	0	1
2	I15013647	O15029016	13	F	1	1	0	0	0	1	0	0	0	0	1	0	1	1	0	0	1	1	1	2	1	1	1	1	1	1	0	0	0	1
3	I15017263	O15028434	10	M	1	0	0	0	0	0	0	0	1	0	0	0	1	1	0	0	1	2	1	2	1	0	1	1	1	1	1	1	0	1
4	I15016356	O15035867	14	M	1	1	0	0	0	0	0	0	0	0	0	0	0	1	0	0	1	1	2	2	1	0	1	1	1	1	0	0	0	0
5	I13022477	O11082542	12	F	1	0	1	0	0	0	0	0	0	0	1	1	1	1	0	0	1	2	2	2	1	1	1	1	1	1	0	0	0	0
6	I14003619	O14009209	9	M	1	1	0	0	1	0	0	0	0	0	0	0	1	1	0	0	1	2	1	2	1	1	1	1	1	1	0	0	1	1
7	I12028087	O12071001	13	F	1	0	0	0	0	0	0	0	0	1	0	0	1	1	1	0	1	2	2	1	1	0	1	1	1	1	0	0	0	1
8	I12022025	O12055112	14	F	1	1	0	1	0	0	0	1	0	0	0	0	0	1	0	1	1	1	1	2	1	1	1	1	1	1	1	1	0	0
9	I14001203	O14003193	15	M	1	1	1	0	0	0	0	0	0	0	1	1	1	1	0	0	1	2	2	2	1	1	1	1	1	1	0	0	0	1
10	I13037900	O08002145	11	F	0	1	0	0	1	0	0	0	0	0	0	0	1	1	0	0	1	1	1	2	1	1	0	1	1	0	0	0	1	0
11	I12001273	O12000748	15	M	1	0	0	0	0	0	0	0	0	0	0	0	1	1	0	0	1	1	1	2	1	1	1	1	1	1	0	0	0	1
12	I12037285	O12092670	14	F	1	1	0	1	0	0	0	1	0	0	0	0	1	1	0	1	1	1	1	2	1	1	1	1	1	1	1	1	0	0
13	I14032692	O14078180	15	F	1	1	0	0	0	0	0	0	0	0	0	0	0	1	0	0	1	1	2	2	1	0	1	1	1	1	0	0	0	1
14	I15013431	O14020279	11	M	1	0	0	0	0	0	0	0	0	0	0	0	1	1	1	0	0	0	0	2	1	0	1	1	1	1	0	0	0	1
15	I13009159	O13021309	12	F	1	1	1	0	0	0	0	0	0	0	0	1	1	0	0	0	1	1	2	2	1	1	1	1	1	1	0	0	0	0

0 - No

1 - Yes

2 - Not done.